The Organizing Committee would like to thank Dean and Staff of the Faculty of Biology, Faculty of Physics, Faculty of Psychology, College of Inter-Faculty Individual Studies in Mathematics and Natural Sciences of the University of Warsaw for their financial and scientific support.

Conference was also financially supported by University of Warsaw Foundation, Universitatis Varsoviensis Foundation and The University of Warsaw Students’ Union.
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Dear Colleagues,

It is our great pleasure to welcome you to the International Conference “Aspects of Neuroscience”. This is the sixth edition of our conference and for the fourth time we organize it at the international level.

We gathered here, because of one, both common and obvious reason: neuroscience. This meeting between outstanding scientists and ambitious young researchers is a great opportunity to fulfill our curiosity.

Interdisciplinary character of this conference is a base for integration of diverse scientific environments in order to create a new quality in brain research. We would like to emphasize that lectures and discussions during our conference, beside undoubted scientific value, are also a perfect chance to establish new ideas and prospective cooperation.

Therefore, we warmly encourage you to participate actively in every aspect of this meeting. We believe that inspirations found during our conference could grow to a new way of thinking in the near future and lead to even greater acceleration in international brain research.

Kind regards,

Organizing Committee

Visit our website: www.neuroaspects.org
ORGANIZING COMMITTEE

The Conference is organized by members of Neurobiology Scientific Student Association at University of Warsaw.

Head of the Conference
Klaudia Jączyńska

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| Marta Królak      | Maciej Winiarski |
| Aleksandra Krysiak| Maja Wójcik |
| Shur Kucman        | Oliwia Zaborowska |

under supervision of

Piotr Borsuk, PhD, Deputy Dean for Studies and Students Affairs of Faculty of Biology

Magdalena Markowska, PhD, Department of Animal Physiology, Faculty of Biology, University of Warsaw; Scientific tutor of Neurobiology Student’s Scientific Club, Warsaw, Poland

Jan Jabłonka, PhD, Department of Animal Physiology, Faculty of Biology, University of Warsaw; Scientific tutor of Neurobiology Student’s Scientific Club, Warsaw, Poland

The Organizing Committee would like to commemorate our dear colleague – Piotr Bogdanowicz. We are grateful for his contribution to development of the Conference Aspects of Neuroscience. Tragically, Piotr passed away in the mountains in Georgia in 2014.
HONORARY PATRONAGES
Ministry of Science and Higher Education
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Prof. Andrzej Wróbel,
Laboratory of Visual System, Department of Neurophysiology, Nencki Institute of Experimental Biology PAS, Warsaw, Poland
Aspects of Neuroscience 2016 Conference Program

November 25, 2016 (Friday)

10.30 Registration opens
11.00–17.30 Workshops
18.00 Opening ceremony

Opening lecture
18.15–19.15 (audience hall 9B)
The transition to minimal consciousness through the evolution of associative learning
Prof. Eva Jablonka

20.00 City Tour

November 26, 2016 (Saturday)

09.00–11.05 I SESSION (Neurobiology)
09.00–10.00 (audience hall 9B)
Molecular Mechanisms of Forebrain Development
Prof. David Price

10.05–11.05 I seminar session

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11.20–12.20 (audience hall 9B)
Practical Experiments into Neural Technology
Prof. Kevin Warwick

12:20–13.20 II seminar session (audience hall 9B)

<p>| 12.25–12.45 Modelling Changes in Implicit Racial Bias after an Immersive Virtual Reality Experience | |
| Rachel Bedder | |
| <strong>12.45-13.05</strong> Extracellular potassium and focal seizures – insight from in silico study | |
| Damiano Gentiletti | |
| <strong>13.05-13.25</strong> Parcellation of the human amygdala: A resting - state fMRI approach | |
| Krzysztof Bielski | |</p>
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Closing lecture
13.40–14.50 (audience hall 9B)
The Emergence of a Circuit Model for Addiction
Prof. Christian Lüscher

14.50   Awards Ceremony and Closing Remarks
Eva Jablonka
Session: Opening lecture
Institute: The Cohn Institute for History and Philosophy of Science and Ideas, Tel Aviv University, Israel
Biography: Eva Jablonka has a M.Sc. in Microbiology from Ben-Gurion University, Israel and a Ph.D in Genetics from the Hebrew University, Jerusalem, Israel. Her postdoctoral studies were in the Philosophy of Science, and in Developmental Genetics. She is a professor in the Cohn Institute for History and Philosophy of Science and Ideas, Tel-Aviv and a member of the Sagol School for Brain Research. Her main interest is the understanding of evolution, especially evolution that is driven by non-genetic hereditary variations, and the evolution of nervous systems and consciousness. She has published many papers and co-authored several books on these topics.

Abstract: The transition to minimal consciousness through the evolution of associative learning (with Simona Ginsburg and Zohar Bronfman)
The minimal state of consciousness is sentience. This includes any sensory experience—exteroceptive, such as vision and olfaction; interoceptive, such as pain and hunger; or proprioceptive, such as the sense of bodily position and movement. We propose unlimited associative learning (UAL) as the marker of the evolutionary transition to sentience, its phylogenetically earliest manifestation and the driver of its evolution. We define and describe UAL at the behavioral and functional level and argue that the structural-anatomical implementations of this mode of learning in different taxa entail subjective feelings (sentience). We close with a discussion of the evolutionary and ethical implications of our proposal.

David Price
Session: Neurobiology
Institute: Centre for Integrative Physiology, University of Edinburgh, United Kingdom
Biography: I studied Medicine at Edinburgh University and did a PhD at Oxford University on the developmental plasticity of the visual system. I moved to the University of California at Berkeley to do a postdoctoral fellowship on development of invertebrates. I was appointed to Edinburgh University as a Lecturer and promoted to Professor in 2003. I have written two books, one on cortical development (Mechanisms of Cortical Development, with David Willshaw, a theoretician) and one called Building Brains: An Introduction to Neural Development with three colleagues with whom I teach at Edinburgh (published by Wiley in 2011).
Abstract: Molecular Mechanisms of Forebrain Development

The brain contains two major classes of neuron: (1) excitatory neurons, which transmit signals between cells by activating the neurons they connect to; (2) inhibitory neurons, which help keep the excitatory neurons in check by dampening down their activity. Both types are needed in the correct numbers for the brain to work normally. As well and doing different things, excitatory and inhibitory neurons look different and have different chemistries. These differences are created because, early in their existence, each type activates its own distinct set of genes that specify the way in which it develops. We have evidence implicating a protein called Pax6 in the high-level control of this specification process. Pax6 is a transcription factor, meaning that it binds to DNA at specific sites and controls many other genes. We have new data indicating that one of Pax6’s most important functions during early embryonic development of the cerebral cortex is to prevent the activation of genes that would turn cells into inhibitory neurons and promote the activation of genes that would turn cells into excitatory neurons.

In mouse, 70-80% of neurons in mature cerebral cortex are excitatory. They are generated within the cortex from cells that contain high levels of Pax6. The other 20-30% are inhibitory. In normal development, they are generated outside the cortex by cells that express low or no Pax6 and they then migrate into the cortex. The cortex does not normally make its own inhibitory neurons. We have discovered that if Pax6 is removed specifically from cortical cells in the embryo, when they are just starting to make cortical neurons, these cells undergo a highly abnormal, rapid and powerful activation of genes that would be expected to promote the development of inhibitory neurons. In other words, they respond to Pax6 removal by altering their program of gene activation from a pro-excitatory to a pro-inhibitory one. We are currently investigating the mechanisms by which Pax6 controls the balance between these two major cell types and the consequences of misregulation.

Kevin Warwick

Session: Computational Neuroscience
Institute: Coventry University, United Kingdom
Biography: Kevin Warwick is Deputy Vice Chancellor (Research) at Coventry University, England. His main research areas are artificial intelligence, biomedical systems, robotics and cyborgs. Due to his research as a self-experimenter he is frequently referred to as the world’s first Cyborg. Kevin was born in Coventry, UK and left school to join British Telecom. He took his first degree at Aston University, followed by a PhD and research post at Imperial College London. He held positions at Oxford, Newcastle, Warwick and Reading Universities before joining Coventry. Kevin is a Chartered Engineer who has published over 600 research papers. His experiments into implant technology led to him being featured as the cover story on the US magazine, ‘Wired’. He achieved the world’s first direct electronic communication between two human nervous systems, the basis for thought communication. Another project extended human sensory input to include ultrasonics. He also linked his nervous system with the internet in order to control a robot hand directly from his neural signals, across the Atlantic Ocean. He has been awarded higher doctorates (DSc) by Imperial College and the Czech Academy of Sciences, Prague. Kevin has been awarded Honorary Doctorates by 8 UK Universities and one from Saints Cyril & Methodius University, Skopje. He received The IEE Senior Achievement Medal, the IET Mountbatten Medal and the Ellison-Cliffe Medal from the Royal Society of Medicine. In 2000 Kevin presented the Royal Institution Christmas Lectures.
Abstract: Practical Experiments into Neural Technology

In this presentation a practical look is taken at how the use of implant and electrode technology can be employed to create biological brains for robots, to enable human enhancement and to diminish the effects of certain neural illnesses. In all cases the end result is to increase the range of abilities of the recipients. An indication is given of a number of areas in which such technology has already had a profound effect, a key element being the need for a clear interface linking a biological brain directly with computer technology. The emphasis is placed on experimental scientific studies that have been and are being undertaken and reported on. The area of focus is notably the need for a biological/technological connection, where a link is made directly with the cerebral cortex and/or nervous system. The presentation will consider the future in which robots have biological, or part-biological, brains and in which neural implants link the human nervous system bi-directionally with technology and the internet.

Giovanna Mallucci

Session: Clinical Neuroscience
Institute: Department of Clinical Neurosciences, University of Cambridge, United Kingdom
Biography: Giovanna Mallucci is Professor of Clinical Neurosciences at the University of Cambridge and an Honorary Consultant Neurologist at Addenbrooke's Hospital, specialising in Dementia. Her undergraduate degrees were in Physiological Sciences and Medicine from the University of Oxford, with clinical training at University College, London. She obtained her PhD from London University in Neurogenetics, for which she generated the first adult-onset mouse model of prion protein knockout that paved the way to her discoveries about reversibility of early neurodegeneration and underlying mechanisms. Since her PhD she has combined clinical work and basic research and led groups in the MRC Prion Unit (2001-2008) and the MRC Toxicology Unit (2008-present) before moving to Cambridge. Her lab is pioneering interventions targeting common pathways for treatment of dementia. She has received numerous national and international awards for her work, including a SciAm50 award for leadership in research as one of the top 50 scientific innovators worldwide. She is an ERC Consolidator Fellow.

Abstract: Neurodegeneration: from molecules to medicines

This talk will discuss my lab's work focusing on the identification of common pathways of disease, relevant across the spectrum of these neurodegenerative disorders. I will discuss both ‘toxic’ processes that can be targeted to prevent neuron death, and also on regenerative processes that can be harnessed for repair at the synaptic level. Both approaches yielded fundamental new insights into mechanisms of neurodegeneration and opened new therapeutic possibilities.

I will discuss how this has led most recently to the discoveries of: 1) the first small molecule able to prevent neurodegeneration and 2) new approaches harnessing endogenous synaptic repair processes for neuroprotection in neurodegenerative disease, both of which are part of a translational programme for new treatments of dementia.
Daniel S. Margulies

Session: Cognitive Neuroscience
Institute: Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany
Biography: Daniel Margulies leads the Neuroanatomy & Connectivity Research Group at the Max Planck Institute for Human Cognitive and Brain Sciences in Leipzig, which investigates cortical organization by mapping patterns of connectivity using a variety of non-invasive methods. He previously worked at NYU and received his doctoral degree from Humboldt University, for which he won the Otto Hahn Medal from the Max Planck Society. Margulies has over 70 publications on a range of topics related to applications of resting-state fMRI, including human cortical organization, cross-species comparative anatomy, and individual differences with respect to behavior and development.

Abstract: Topographic principles of cortical connectivity

Distinct large-scale cortical networks have been well characterized in recent years, but we are still lacking an integrative understanding of how they spatially related to one another. The hypothesis was recently proposed by Buckner and Krienen (2013) that cortical distance from highly differentiated primary sensory areas may determine gradients of specialization. I will present work extending the ‘tethering hypothesis’ by investigating how spatial proximity along the cortical surface relates to functional specialization and cortical morphology. Beginning from the default-mode network (DMN), we observe a functional continuum from the heterogeneous to highly specialized: more distal regions are specialized in either primary sensory or motor functions and more proximal regions are associated with abstract cognition and internally-oriented content. Domain-general systems subserving functions such as attention, working memory, and executive control are then situated between these two expanses of cortex. The implications of these findings for theories of cortical development and function will be discussed.

Christian Lüscher

Session: Closing lecture
Institute: Department of Basic Neurosciences, University of Geneva, Switzerland
Biography: Christian Lüscher is a professor of Neuroscience at the University of Geneva and an attending in Neurology at the University Hospital of Geneva. He obtained his medical degree from the Universities of Lausanne and Berne. After a residency in Neurology he spent three years at UC San Francisco to study synaptic plasticity. With a career development award from the Swiss National Science Foundation he established his lab at the University of Geneva in 1999 and became full professor in 2009. The Lüscher lab studies the synaptic mechanisms that underlie the behavioural adaptations in drug addiction. Over the last 15 years he has characterised several forms of “drug-evoked synaptic plasticity” and established links of causalities with various components in addiction models. The lab was the first to show that normalising synaptic transmission in vivo with optogenetic approaches can erase pathological behaviour. The group is currently working towards translational protocols to emulate optogenetic protocols with methods of neuromodulation approved for human use such as deep brain stimulation. Christian Lüscher has received several awards, including an ERC advanced grant, the Bing Prize and the Cloëtta Prize.
Abstract: The Emergence of a Circuit Model for Addiction

Addiction is a disease of altered behavior. Addicts use drugs compulsively and will continue to do so despite negative consequences. Even after prolonged periods of abstinence, addicts are at risk of relapse, particularly when cues evoke memories that are associated with drug use. Rodent models mimic many of the core components of addiction, from the initial drug reinforcement to cue-associated relapse and continued drug intake despite negative consequences. Rodent models have also enabled unprecedented mechanistic insight into addiction, revealing plasticity of glutamatergic synaptic transmission evoked by the strong activation of mesolimbic dopamine—a defining feature of all addictive drugs—as a neural substrate for these drug-adaptive behaviors. Cell type-specific optogenetic manipulations have allowed both identification of the relevant circuits and design of protocols to reverse drug-evoked plasticity and to establish links of causality with drug-adaptive behaviors. The emergence of a circuit model for addiction will open the door for novel therapies, such as deep brain stimulation.
WORKSHOPS

This year Aspects of Neuroscience Conference starts with workshops taking place on Friday 25th. Four different workshops to improve your soft skills and broaden methods and tools.

- Eco-HAB & IntelliCage - fully automatically devices for behavioral testing
- First steps with data analysis in Python
- Science communication contests: why, how, what
- Soft skills in science

ECO-HAB & INTELLICAGE - fully automatically devices for behavioral testing

**Time:** 11am - 1pm and 14pm - 16pm, 2 hours each  
**Place:** Laboratory of Neurobiology of Emotions, Nencki Institute of Experimental Biology  
**Trainers:** Maciek Winiarski, Alicja Puścian, Laboratory of Neurobiology of Emotions, Nencki Institute of Experimental Biology

Classical assays for behavioral testing has played a key role in neuroscience. However, they are time-consuming and sensitive to small changes in the testing environment. Nowadays technology allows to develop fully automated devices that replace classical assays. For example, IntelliCage was developed to test cognitive skills (like learning), whereas Eco-HAB allows for testing social behaviors (e.g. social preference).

Workshop will be divided into theoretical and practical part. During the theoretical part we will introduce to you the principles of behavioral testing and explain how to design and prepare automated experiments. During the practical part you will have an opportunity to visit working devices in Nencki Institute of Experimental Biology. You will learn how to assemble Eco-HAB and IntelliCage systems and how to use specialized software to control cages and collect data.

We would like to acknowledge dr Ewelina Knapska - Head of the Laboratory of Neurobiology of Emotions and member of Scientific Committee of the Conference Aspects of Neuroscience for enabling organizing workshops.

**FIRST STEPS WITH DATA ANALYSIS IN PYTHON**

**Time:** 2pm - 5pm, 3 hours  
**Place:** Faculty of Biology  
**Trainer:** Piotr Migdal, PhD

Do you want to learn how to write scripts, and use programming to solve practical data analysis problems? Python is the way to go! It starts in a simple way, but is one of the best environments for data science (including machine learning and artificial neural networks). You will learn how to load data, process it and make plots. It is a basic introduction, starting from basic structures (like dictionaries and lists) and control flow (if, else, loops). If you are clicking your way to get results, or not yet comfortable it any programming language (but would love to change that!), it is for you!

We will be using Jupyter Notebook – an environment, which makes interaction with data convenient and beautiful. Notebooks are also an easy ways to share data analysis and results with others.
SOFT SKILLS IN SCIENCE

**Time:** 4pm – 5.30pm, 1.5 hour  
**Place:** Faculty of Biology  
**Trainer:** Rafał Czajkowski, PhD

This workshop will focus on techniques that might prove helpful when preparing for public talks. The topics include: types of scientific talks and their goals, presentation structure, slide layout dos and don'ts, talk time management.

SCIENCE COMMUNICATION CONTESTS: WHY, HOW, WHAT

**Time:** 1.30pm – 3.30pm, 2 hours  
**Place:** Faculty of Biology  
**Trainer:** Alek Klemba, PhD student at University of Warsaw (MISMaP College)

Have you ever wondered how to explain your research to your family and friends in the way they would understand and enjoy it? Have you ever wanted to share your knowledge with larger audience? If so, this workshop is for you as it will cover basics of science communication and popular science presentation. During the presentation participants will get insight into why and when is it worth to engage in such activity, moreover it will be great opportunity for you to consider pros and cons of taking part in it. You will also learn about the contests and associated trainings (e.g. FameLab and Masterclass). In the practical part, you will draft & deliver your own short popular science presentation (1 – 2 minutes), receiving feedback afterwards.

CITY TOUR

Within the conference fee, participants are given possibility to participate in a city tour. After the opening lecture on Friday we offer you professional Warsaw sightseeing covering famous spots of our capital city.

when: Friday, 8:00pm  
where: Faculty of Biology (and then we transfer to the Old Town)

DISCUSSION

After the end of Saturday’s official part we invite you for the “Questions and Answers” meeting about career opportunities for young scientists. You will have a unique opportunity to ask our guests and find out “How to survive in science”. Feel free to participate actively in discussion.

when: Saturday, 6:30pm  
where: Faculty of Biology

INTEGRATION PARTY

We encourage all participants to take a part in the integration party organized on Saturday. Chill after Saturday science fever, meet the speakers, organizers and students from other countries, give a try to some local beers. Be prepare for a little neuronerdy surprise. It’s gonna be fun, for sure!

when: Saturday, 9:00pm  
where: “Solec 44” Restaurant. Address: Solec 44, Warsaw
ORAL PRESENTATIONS - NEUROBIOLOGY

Noradrenergic modulation of medial prefrontal cortex (mPFC) pyramidal neurons.
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Noradrenaline (NA) is an important factor in the regulation of cognitive brain functions and affective processes. The medial prefrontal cortex (mPFC) receives dense noradrenergic projections from locus coeruleus. Impaired modulation of PFC by NA has been implicated in widespread neuropsychiatric diseases such as posttraumatic stress disorder, attention deficit hyperactivity disorder, and depression. The aim of our study was to investigate which adrenergic receptor subtypes are the main target of action of NA in mPFC pyramidal neurons and what are the cellular mechanisms underpinning the effects of NA in these neurons. To answer the questions, we have recorded the resting membrane potential and holding currents using patch-clamp techniques. Gramicidin perforated-patch and classical whole-cell recordings were obtained from layer V mPFC pyramidal neurons in slices isolated from young rats. Tested compounds were applied to the bath and/or to the solution in the recording pipette. Obtained results showed that activation of \( \alpha_1 \) and \( \alpha_2 \)-receptors did not significantly affect the membrane potential in mPFC pyramidal neurons. Meanwhile, application of NA evoked dose-dependent depolarization of membrane potential and inward currents. The effects were considerably reduced by the nonselective \( \alpha \)-receptor antagonist propranolol and the selective \( \alpha_1 \)-receptor antagonist metoprolol. Moreover, the nonselective \( \alpha \)-receptor agonist isoproterenol, the \( \alpha_1 \)-receptor agonist dobutamine and the selective \( \alpha_3 \)-receptor agonist BRL37344 mimicked the inward currents caused by NA. We conclude that NA modulates mPFC pyramidal neurons acting via \( \alpha_1 \) and \( \alpha_3 \)-receptors, causing changes in the membrane potential and the holding currents. Our data suggest that activation of the \( \alpha_3 \)-receptor evokes inward current due to HCN channel activation. The effect is probably mediated by the phospholipase C signaling pathway and does not involve adenylyl cyclase. Supported by National Science Centre, Poland, grant 2014/15/N/NZ4/04760 and FW5/PM2/16.

Synaptic potentiation at basal and apical dendrites of hippocampal pyramidal neurons employs divergent postsynaptic signaling pathways

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In the central nervous system, experience-dependent plasticity, learning and memory require the activity-dependent control of synaptic efficacy. However, the molecular mechanism of circuit-specific plasticity remain not fully understood. In particular, CA1 hippocampal pyramidal neurons receive multiple inputs to basal and apical dendritic trees that carry different amount of spatial and non-spatial information. While synaptic plasticity at apical dendrites has been intensively investigated, the molecular mechanism of synaptic gain at basal dendrites received less attention. Here, we combined electrophysiology and pharmacology to study intracellular cascades involved in AMPAR- and NMDAR-mediated synaptic gain in CA1 pyramidal neurons dendritic trees. To this end, we recorded local field potentials at apical dendrites (stratum radiatum, SR) or basal dendrites (stratum oriens, SO) in response to stimulation of afferents located in the same plane in acute brain slices of adult P45-60 C57BL/6 mice. We found that in response to HFS (high frequency
stimulation protocol, 4x100Hz, every 10s), SR and SO synapses underwent long-term potentiation (LTP) of AMPAR- and NMDAR-mediated synaptic transmission that were similar in magnitudes. Moreover, LTPAMPA in both SR and SO exhibited a similar magnitude in response to multiple HFS applied every 15 minutes and was completely abolished in the presence of NMDAR-antagonist APV (50μM) indicating that both hippocampal projections expressed canonical form of LTP. We next analyzed in more detail the NMDAR-component of synaptic responses. We found that HFS resulted in LTPNMDA in SR and SO that was equally sensitive to protein kinase A inhibitor (H89; 10μM). However LTPNMDA at SO was insensitive to wide-spectrum RhoGTPases inhibitor (rhosin; 20μM) unlike SR synapses. In addition, administration of dopamine receptor agonist ((R)-(+)−SKF-38393 hydrochloride;1μM, D1-like dopamine receptor selective partial agonist) led to LTPAMPA and LTPNMDA exclusively in SO but not SR synapses. Altogether, we report that excitatory synapses located on neighboring dendritic trees undergo gain in synaptic function following enhanced afferent activity but employ a divergent set of molecular cues. We hypothesize that promotion of LTPAMPA and LTPNMDA at basal dendrites may promote associative and spike-timing dependent plasticity at apical dendrites and enhance sparse information encoding at CA1 hippocampal region.

The role of central amygdala in social transmission of fear
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In many animal species, emotional states can be transferred between individuals through observation. This process, known as emotional contagion, is a basic form of communication and is probably prerequisite for development of higher-order social skills (such as empathy). In spite of many years of research on emotions, the transmission of affective states between subjects is poorly understood. In order to study this phenomenon, we used an experimental model in which one rat (observer) is watching a cage-mate (demonstrator) undergoing contextual fear conditioning. Our behavioural results indicate this kind of stimulation promotes communication between animals with 22-kHz ultrasonic-vocalizations, which are known to occur in aversive situations. What is more, observer rats mimic the behaviour of demonstrators and display robust freezing. We hypothesized that social transmission of fear depends on central nucleus of the amygdala (CeA), a brain structure which is well known to be engaged in fear conditioning in individual animals. To check this hypothesis, we injected a viral vector carrying channelrhodopsin 2 (ChR2) sequence into CeA. To ensure that the ChR2 would be expressed only in the subpopulation of CeA cells which are activated by the observational fear, it was placed under the c-fos gene promoter. Two weeks after the surgery the rats underwent observational transfer of fear; the control group was exposed to the experimental cage for the same amount of time. 24 hours later the rats were put into modified version of open field and the cells which had expressed c-fos on the previous day were activated with blue light. The stimulation resulted in robust increase in avoidance behaviours in the experimental group, strongly suggesting that CeA cells which are activated by observational fear regulate anxiety. To study the functional connectivity of CeA cells during social transmission of fear, we used transgenic Venus-PSD95 rats in combination with PHA-L anterograde tracer. Preliminary results suggest that within many brain structures, the majority of cells which were activated by observational fear were getting projections from CeA. To sum up, the results indicate that CeA circuit activated by social transfer of fear regulate defensive behaviours, probably through their extensive connections with major neuromodulatory systems.
How to build a tree: the function of Angiomotin family of proteins in the brain
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Dysfunction in synaptic transmission causes many disorders such as epilepsy, schizophrenia or depression. Therefore understanding of molecular processes that govern organization of the neuronal networks is in the center of attention of the neurobiological research. In this study we investigated the expression and function of Angiomotin family of proteins comprising of Amot, Amotl1 and Amotl2 in the central nervous system (CNS). We have discovered that all three proteins are expressed in neurons where Amotl1 and Amotl2 localize to the synaptic compartments and Amot is distributed in axons and dendrites. Our functional experiments on cultured neurons revealed that Amot regulates dendritic tree arborization. Using protein complex purification from neurons combined with mass-spectrometry protein analysis, we identified several Amot-interacting proteins including YAP, a component of the Hippo signaling pathway and Coronin2b that belongs to a family of actin organizing proteins, which were responsible for dendritic development. To study the function of Amot in vivo we generated conditional knockout mice with neuron-specific deletion of Amot. Mutant mice had abnormal cerebellar morphology and also exhibited defects in motor coordination, as studied in behavioral tests. To analyze single neuron morphology in vivo we used two methods to label sparsely individual cells in the brain: (1) we infected neurons with low titer of GFP-expressing AAV viruses, and (2) labeled neurons using inducible recombination combined with fluorescent labeling of mutant cells (CRE-LoxP system with tamoxifen induced deletion of Amot and STOP cassette in front of Tomato reporter). Interestingly, deletion of Amot in neurons led to the abnormalities in the development of the dendritic tree, similarly to the phenotype observed in vitro. In contrast to Amot mutant mice, neuronal deletion of Amotl1 did not affect motor coordination, but led to severe defects in the ability of mice to build nests, suggesting abnormalities in social behavior. Collectively, our research identified a novel family of protein that regulate neuronal organization and behavior of living animals. This research was supported by the NCN grants Sonata-Bis 2012/05/E/NZ3/00487, Preludium 2015/19/N/NZ3/02346, and Opus 2014/13/B/NZ3/00909.

The study of the molecular basis of fragile X syndrome
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Neuroligins (NLGNs) are postsynaptic adhesion molecules that bind to presynaptic neurexins what is crucial for functional synapses formation in the brain. It has been shown that mutations in Nlgn3 and Nlgn4 genes are associated with autistic phenotype. There is also a known single gene cause of autistic behaviors: fragile X syndrome (FXS). This condition results from the lack of fragile X mental retardation protein (FMRP). In physiological conditions FMRP by binding to neuronal mRNAs regulates the local translation of transcripts that play an important role in synaptic signaling and plasticity. The aim of this project was to determine whether the synaptic translation of Nlgn3 mRNA is regulated by FMRP. We used Fmr1 knock-out mice – the model of FXS and synaptoneurosomes
– the model of isolated synapses, which retain the ability to respond to stimulation in vitro. We have shown that Nlgn3 mRNA immunoprecipitates with anti-FMRP antibody what suggests that Nlgn3 mRNA associate with FMRP in synaptoneurosomes. To confirm this finding, we investigated the colocalization of Nlgn3 mRNA and FMRP in hippocampal neurons using the fluorescence in situ hybridization method. At the same time, the Western Blot analysis revealed that the level of NLGN3 is elevated at Fmr1 KO synapses. What is important, the qRT-PCR analysis showed that there is no difference in synaptic level of Nlgn3 mRNA between wild-type and Fmr1 KO mice. The identification of deregulated local translation of Nlgn3 mRNA in Fmr1 KO mice led us to investigate the time-dependent dynamics of surface and intracellular NLGN3 protein distribution at the synapses upon stimulation. The chemical crosslinking method revealed that Fmr1 KO synapses remain able to respond to stimulation and the synaptic plasticity is not fully impaired in fragile X mice. To conclude, gathered results indicate that local synaptic translation of Nlgn3 mRNA is regulated by FMRP what leads to elevated amounts of NLGN3 protein at Fmr1 KO synapses.
Modelling Changes in Implicit Racial Bias after an Immersive Virtual Reality Experience

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Several studies have demonstrated that embodiment in a dark-skinned avatar during immersive virtual reality (IVR) can reduce implicit racial bias in light-skinned participants, as measured by Implicit Association Test (IAT) scores. However, mechanistic explanations of this effect are still largely speculative. Here, we present a neural network model which hypothesises that the observed reduction in implicit bias is due to assimilation of the dark-skinned virtual body into the participant’s self-image. The model consists of an auto-associative network with neurons that are tuned to respond to perceived self-image features / IAT stimuli (e.g. dark-skinned faces, positive words). The network learns an initial self-image which can then be updated by an IVR experience. The network output is used as sensory input to a drift diffusion model of the IAT. We demonstrate that the model can replicate the results of two recent behavioural IVR experiments. In addition, the model predicts that the magnitude of changes in implicit bias should correlate with measures of self-esteem. We conclude that a reduction in implicit racial bias in light-skinned participants is caused by the dark-skinned features being perceived as more similar to the participant’s self-image after embodiment. This finding raises the intriguing possibility of reducing implicit bias with virtual reality video-gaming.

Extracellular potassium and focal seizures – insight from in silico study

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It is generally considered that epilepsy and seizures are related to alteration in neuronal excitation/inhibition balance. However, using an in vitro isolated guinea pig brain model of focal seizures it has been shown that seizures were initiated with increased firing of inhibitory interneurons, neuronal silence of principal cells and increase of extracellular potassium concentration. Neuronal firing of principal cells was subsequently restored with acceleration-deceleration firing pattern followed by rhythmic burst activity. In order to investigate the link between ionic dynamics and the experimentally observed seizure pattern we developed a computational model of hippocampal cells embedded in the extracellular space with realistic dynamics of Na⁺, K⁺ and Cl⁻ ions, the glial uptake system and diffusion mechanisms. We show that ion concentration changes exert significant influence on the network behaviour. In particular, we show that in the model, strong discharge of inhibitory interneurons may result in long lasting accumulation of extracellular K⁺, which sustains depolarization of principal cells and causes their pathological discharges. This effect is not present in a reduced model with fixed ionic concentrations. Using the model, we investigated the main sources of extracellular K⁺ accumulation and the role of various components of potassium homeostasis. Finally we suggest a novel antiepileptic therapy targeting the potassium regulation mechanism. Such an innovative strategy could be possibly accomplished by developing a nanoparticle potassium
buffering system. Model simulations show that such artificial pharmacological agents present in the extracellular space have anticonvulsant effects. This novel therapeutic strategy could provide new avenues for successful seizure control.

**Parcellation of the human amygdala: A resting-state fMRI approach**

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The amygdala is a heterogenous, subcortical structure located bilaterally in the medial temporal lobes. Animal studies indicate that groups of nuclei situated in different parts of the amygdala are components of distinct neural circuits underlying emotional and cognitive processes. Based on post-mortem analyses, the human amygdala is divided into three main subdivisions: basolateral, centromedial and superficial (cortical). Combining this results with standard MNI152 space in Julich Probabilistic Atlas allows to study functional properties of distinct parts of human amygdala in vivo. However, increasing number of studies provides evidence that this method does not recognize different subdivisions in individual subjects reliably. For that reason, non-invasive parcellation schemes for human amygdala are developed. The proposed methods include manual segmentation of structural MRI images, probabilistic tractography of diffusion tensor imaging (DTI) or functional connectivity studies during resting-state using functional resonance imaging (rsfMRI). Our main aim is to develop an approach for individual parcellation based on rsfMRI data. We have used dense connectome (group averaged data from 468 subjects) derived from Human Connectome Project and performed hierarchical clustering to group amygdala's voxel into clusters with similar connectivity patterns. Results suggest that it is possible to divide human amygdala into four subdivisions: basal, lateral, centromedial and cortical. Obtained subdivisions correspond to anatomical parts in respect of size, localization and connectivity patterns. This parcellation seems to be compromise between sensitivity of the statistical procedure and spatial resolution of fMRI parameters. The next goal is to assess the reliability of the method for individual parcellations. This study was supported by a grant (2014/15/B/HS6/03658) from the Polish National Science Center.
Electrical stimulation of vagus nerve as a promising tool in neurodegenerative disorders treatment
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Electrical stimulation in vagus nerve (VNS) is under the investigation as a promising treatment for several psychiatric and neurological disorders like: Alzheimer disease, drug-resistant epilepsy, schizophrenia, migraine or central inflammation. VNS was also described to potentiate hippocampal LTP in rats as well as cognitive function and memory processes in humans. In the present study different stimulation parameters of VNS were evaluated in order to induce hippocampal formation (HPC) theta rhythm type field potential in anesthetized Wistar rats. Animals were implanted with a VNS bipolar electrode around the left vagus nerve. Tungsten recording electrode was implanted in the left dentate gyrus for EEG field potential. Various square pulse parameters were tested: pulse duration (1–1.5 ms), train duration (10s), frequency (10–60 Hz) and intensity (0.2–10 mA). Vagus nerve stimulation was done once every 5, 10, 30 and 60 min. Different parameters of theta rhythm type II were analyzed before and after VNS. We demonstrated the first time the presence of HPC type II in response to the application of VNS. Depending on the different experimental protocols, theta could occur in the same time as electrical stimulation of vagus nerve (brief effect) or after this procedure (delayed effect). Positive VNS effect on hippocampal theta field potential may suggest the involvement of vagus nerve excitation in number of central functions related with a presence of theta including improvement in sensory-motor integration, increase in cognitive functions and memory processing.

Ceftriaxone and N-acetylcysteine as a neuroprotective strategies in brain ischemia.
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Glutamate (Glu) plays a key role in excitotoxicity-related injury in cerebral ischemia. In the brain, Glu homeostasis depends on Glu transporters, including the excitatory amino acid transporters and the cysteine/Glu antiporter (xc−). We hypothesized that drugs acting on Glu transporters, such as ceftriaxone (CEF, 200 mg/kg, i.p.) and N-acetylcysteine (NAC, 150 mg/kg, i.p.), administered repeatedly for 5 days before focal cerebral ischemia in rats and induced by a 90-min middle cerebral artery occlusion (MCAO), may induce brain tolerance to ischemia. We compared the effects of these drugs on brain infarct volume, neurological deficits and protein expression of the Glu transporter-1 (GLT-1) and xc− with the effects of ischemic preconditioning and chemical preconditioning using 3-nitropropionic acid (reference preconditioning strategies). Moreover, we performed microdialysis of two brain structures – frontal cortex and hippocampus and in the obtained aCSF we determined the fluctuations of glutamate before, during and after middle cerebral artery occlusion. Our results
indicate that administration of CEF and NAC significantly reduced infarct size and neurological deficits caused by a 90-min MCAO. These beneficial effects were accompanied by changes in GLT-1 protein expression caused by a 90-min MCAO in the frontal cortex and hippocampus. These changes correlated with the concentration of glutamate, not only during ischemia onset, but also after recirculation. Our results suggest that the regulation of GLT-1 and xc- plays a role in the development of cerebral tolerance to ischemia and that this regulation may be a novel approach in the therapy of brain ischemia.

**Heligmosomoides polygyrus infection in EAE mice causes higher leukocytes migration into CSF and nervous tissue regeneration**

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Introduction: Both epidemiological and laboratory data suggest that helminth infection may have beneficial effect on patients with autoimmune diseases. Inflammation inhibition and symptoms reduction is often observed. Infection with Necator americanus or Trichuris suis is considered as an alternative treatment for patients suffering of Inflammatory Bowel Disease or Multiple sclerosis. The efficiency of treatment is verified in 2nd and 3rd stage clinical trials. Nonetheless mechanisms of helminth immune regulation remain unclear. Infection with the murine intestinal nematode Heligmosomoides polygyrus, established model of immunomodulation, reduces experimental autoimmune encephalomyelitis (EAE) symptoms. The parasite L4 stage larvae meaningly down-regulate peripheral immune response. Methods: C57Bl6 female micewere studied at 6 day post H. polygyrus infection (dpi) and 28 day post EAE induction. Permeability of blood–brain barrier (BBB) was checked with use of 2% Evans Blue. Leukocytes in CSF were counted with Muse Count and Viability and Diff Quick staining and 8 μm thick frozen brain slides were stained with Eosin & Hematoxylin. Cytokine level in CSF was measured by ELISA test. Results: In EAE mice we observed increase in BBB permeability and greater infiltration with leukocytes during the histotropic phase of the parasite infection, especially on the 3rd day. On the 6th day of H. polygyrus infection in cerebrospinal fluid regulatory cytokine- TGF-β level was increased and pro-inflammatory IL-12 level was decreased. We noticed regeneration and remyelination of nervous tissue. Percentage input of polymorphonuclear cells I and II was changed. This work was supported by Polish National Science Centre Grants No. 97222 and 218668 and 277233.

**Alterations in cell cycle regulation in lymphocytes of patients in the early stage of Alzheimer’s disease precede massive neuronal loss in the brain**

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Alzheimer’s disease (AD) is a progressive insidious dementia that continues to severe debility and finally ends with death. The pathogenesis of AD is still unclear but some symptoms occur earlier than changes observed in brain. Mild Cognitive Impairment (MCI) represents early AD stage, preceding massive deposition of Aβ aggregates and associated neuronal loss in the brain that manifest as a major cognitive decline. Importantly, in AD molecular alterations are observed not only in brain neurons, but also in peripheral cells such as blood lymphocytes. Therefore easily accessible lymphocytes represent an attractive alternative to studies on AD pathomechanisms. Besides, lymphocytes seems
promising as a material for diagnostics and drug screening. Recently, we and others demonstrated that immortalized B lymphoblasts from sporadic AD (SAD) patients show aberrant cell cycle, linked with upregulated levels of p21, the key protein in G1/S cell cycle checkpoint and in the apoptotic response\textsuperscript{1,2}. In the current study we aimed at elucidation if p21 levels are altered early in AD, in lymphocytes of MCI patients, and to investigate the effects of p21 on the apoptotic response of SAD and MCI lymphocytes to oxidative stress (OS) evoked by 2-deoxy-D-ribose (2dRib). We report highly increased levels of p21 protein and associated increase of cells in the G1 phase of the cell cycle in freshly isolated B cells from SAD and MCI patients. In response to an oxidative stress insult evoked by 2-deoxyRibose (2dRib), p21 levels were downregulated in B cells of both MCI and AD patients, in contrast to control cells from non-demented patients as well as from patients with hypertension. These results suggest that changes in the p21 levels and in lymphocytes appear early in AD and gradually increase with the disease progression. Furthermore, they support the hypothesis of an early role of an aberrant cell cycle and of altered response to oxidative stress in the progression of AD pathology. Summarizing, these work highlights the application of blood lymphocytes as potential diagnostic tissue and p21 protein as potential biomarker of early AD.

**Trophic and immunomodulatory characteristics of bone marrow-derived mesenchymal stem cells obtained from children diagnosed with hypoxic-ischemic encephalopathy**

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Mesenchymal stem cells (MSC) are gaining increased interest of regenerative medicine as a tool in cellular therapies of pathological processes in nervous system, driven by excessive inflammation and neurodegeneration. MSC, which originally populate adult and neonatal tissues, have anti-inflammatory and immunomodulatory properties. Moreover, MSC are known to release many trophic factors, that can mediate extensive tissue repair. The results of our recent clinical studies demonstrated that autologous MSC transplantation in children with hypoxic-ischemic encephalopathy (HIE) leads to an improvement of the clinical status of patients. The aim of this study was to evaluate properties of patients’ autologous MSC. Isolated CD271\textsuperscript{+} cells from mononuclear fraction of autologous bone marrow were plated into tissue culture flasks in culture medium. Flasks were incubated in standard culture conditions. The MSC phenotype was analyzed with antibodies for CD3, CD45, CD73, CD90 and CD105. In order to evaluate changes in immunomodulatory and tissue remodeling properties, MSC were additionally seeded on 6-well plate and after reaching 80% confluence, incubated with INF-\textgamma, TNF-\textalpha or IL-1\textalpha. RNA were isolated from MSC in various conditions and reverse transcription was performed. Gene expression was determined by qPCR analysis. Expanded MSCs were positive for CD73, CD90, CD105 and negative for CD3 and CD45. Gene expression analysis have shown, that MSC express genes for neurotrophic (BDNF, GDNF, CNTF, NGF), proangiogenic (FGF-2, VEGFA) factors and also ones related to tissue remodeling (MMP2, HGF). We have also evaluated the changes in immunomodulatory (IDO, ARG) and tissue remodeling (MMP2, MMP9) properties in control conditions and after incubation with proinflammatory cytokines. Obtained results confirmed unique properties of MSC and gave us presumptive evidence to explain their neuroregenerative potential in HIE treatment.
Evidence integration of auditory textures is task-dependent

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Many natural sounds can only be characterized on a statistical level, while their spectro-temporal structure is highly variable, e.g. wind, rain or crackling fire. These ‘acoustic textures’ can be classified easily by humans, despite a substantial amount of variability between examples from the same type. Previous studies have shown that classification of a texture improves with the time the texture can be sampled, but the neural representation underlying this classification has not been addressed. In the present study, we recorded the neural response using EEG in human subjects listening to natural auditory textures that changed their statistics at a random point in time. Subjects were instructed to detect changes and either report (active condition, button press) or not report (passive condition) them. The results indicate that the acoustic stimulus is represented in auditory cortex by a transient response, while the change in statistics is barely detectable. Conversely, in centro-parietal electrodes, the stimulus onset led to a sustained response and the change in statistics leads to a large, late response. This response scaled with the difficulty of the task, i.e. with the time the first texture statistics could be sampled, and the size of the change in statistics. In the passive condition, the response to stimulus onset was the same, however, changes in statistics elicited much smaller responses in the centro-parietal electrodes. In summary, statistical acoustic stimuli, such as auditory textures, are represented in auditory cortex, but the integration of evidence for detecting changes occurs only later in parietal areas. The integration occurs even in the absence of response reporting, however, to a smaller degree, suggesting a possible use of the paradigm in separating different states of consciousness without reporting.

Orthographic representation of tactile Braille words in congenitally blind. Preliminary results of an repetition suppression study

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The way the human brain is organized is one of the key issues in today’s neuroscience. One way to understand its mechanisms better is through the processes involved in reading – a uniquely human ability. It is known that in sighted, the Visual Word Form Area (VWFA) in the left vOT shows an increased activation in response to orthographical analysis of written words. However, reading theories challenge the existence of neural representations that code for the orthographic representation of written pseudo and real words in the sighted. Glezer (et al., 2009, 2015) showed evidence that the human brain contains neurons, found in the VWFA (left vOT), with high selectivity to orthographical representations of both pseudo and real words in the sighted. Recently it was suggested that when reading by touch, the congenitally blind show similar activation in the VWFA to that seen in sighted reading (Reich et al., 2011). Our study set out to test whether the vOT in the blind performs an analogous function to the VWFA in the sighted, by investigating whether the vOT in the blind is sensitive to orthography of Braille words. We tested congenitally blind with
a classic priming paradigm. Participants were exposed to prime-target pairs of pseudowords (same, different, different) in tactile modality. Since the VWFA in the sighted responds less strongly to spoken auditory stimuli, we presented additional control conditions in the auditory modality (same, different) while subjects underwent fMRI. (1) same, in which the same stimulus was presented twice in each trial; 2) 1different, in which the first and second stimulus differed by one letter and; 3) different, in which stimuli shared no letters. The ventral visual system robustly differentiated Braille letter strings, thus replicating our previous findings (Reich, et. al, 2011). Moreover, we found that not only priming effect is evident in such a serial script as Braille, but more importantly that, the rapid adaptation effect for pairs of words that differ is observable in the ventral occipito-temporal cortex of the blind. Crucially, we found no activation in this region in response to the same prime-target stimuli pairs presented in auditory modality which is consistent with the pattern of activation in the sighted VWFA. The results thus support our hypothesis that the vOT in the blind performs a function analogous to the VWFA in the sighted, independently of the sensory input.

The effect of lateralized saccadic eye movements and horizontal optokinetic stimulation on asymmetrical attentive blank stares distribution: An EFRP study

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Attentive blank stares mean a failure to notice changes in a visual scene, despite looking at the area of change. In our previous studies we showed that people may differ greatly in terms of the number of attentive blank stares and that this effect did not disappear even after the high visual working memory group were given expertise training. Moreover, by using EFRP (Eye Fixation - Related Potentials) method it was possible to empirically test the hypothesis that blank stares can be regarded as an example of temporary disengagement between overt and covert attention. In the present EFRP study we tested the effect of leftward/rightward saccadic eye movements (LS/RS) and horizontal optokinetic stimulation (hOKS) on asymmetrical attentive blank stares distribution. On the one hand, previous data show a possible advantage of RS over LS in horizontal shifts of attention. On the other hand, studies on covert shifts of attention showed left, but not right visual field advantage in the change detection task, with the possibility of cross-overs between them. Moreover, there are also data showing the mixed effects of hOKS on attention shifts. Our study involved 30 people (inc. 15 women) between the ages 19-27 years. Results of 5 subjects were excluded from the analysis because of technical problems with the EFRP data recordings. The change detection task was a combination of the standard saccadic task and the flicker task, which allowed to test the number of attentive blank stares just after LS/RS. Subjects performed the change detection task in 3 hOKS conditions: leftward, rightward and no movement (480 trials each); hOKS consisted of exposure - on the periphery of vision - vertical black and white stripes, alternating horizontally. There were less attentive blank stares just after RS than LS (F(1,22)=21.23,p<.001,part.α²=.49). Additionally, we found out advantage of RS over LS, which manifested in responses times reduction, an increase of responses confidence; however, analysis of simple effects showed that these RS advantage manifestations depended on the type of hOKS conditions. Regarding EFRP results, there was a significant differences between the hOKS conditions, but only for the change detections made after RS, but not LS. We discuss our results in the context of previous studies, among others, pointing out the role of left and right hemisphere in asymmetrical distribution of spatial attention.

This study was supported by a grant from the Polish National Science Centre (NCN nr 2013/09/B/HS6/03266; UMO-2013/09/B0HS6/03266)
**Alpha decrease prepare visual system to the forthcoming task**

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When expecting upcoming task, brain turns into attentive state in order to respond with better sensitivity to stimulation. In studies of attention alpha has been reported to decrease over the cortical regions involved in stimulus processing, with the magnitude of modulation correlating with performance. We aimed to verify if the size of alpha desynchronization in the anticipatory period depends on whether the forthcoming task requires attention. The experiment consisted of two contrasting types of trials, one where participants were required to perform a demanding visual search task or second where they executed a pre-determined response. Participants were presented with cue displayed in the central visual field for 2, 3, 4 or 5 sec. In the visual search trials, the cue defined the target for the following visual search task. In the simple response trials, the cue directly encoded proper response. The cues for both conditions were visually identical except for the used color, coding the trial type. The trials were separated with periods during which only fixation cross was present, enabling assessment of baseline activity. EEG was continuously recorded from 62 scalp sites positioned according to the extended 10–20 system. The anticipation period corresponding to the cue presentation was investigated with time-frequency analysis. Statistical evaluation was preformed using repeated measures cluster mass permutation test to deal with multiple comparisons problem in multidimensional data. The decrease of amplitudes in alpha and beta bands was present through entire cue period when compared to fixation. This effect was most prominent over occipital and parietal and to lesser extent over frontal regions. The decrease was greater during anticipation period in the difficult search task than in the simple response condition. Our results indicate that the time preceding actual task execution is actively modified by the top-down processes.

**Cognitive reappraisal of pain - an EEG study**

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Cognitive reappraisal is one of the best studied as well as well-established affective self-regulatory strategies¹. It involves reinterpreting the meaning of emotionally evocative stimuli in order to change one’s emotional response to it. As its efficacy was proved in multiple studies using visual stimulation, we aimed at investigating whether its influences can be generalized to different domains, such as pain. Pain as a type of negative experience was proved to be highly susceptible to diversified forms of top-down regulation². We were particularly interested in the nature of an underlying mechanism of presumed pain modulation. To address this issue, we recorded ERP responses and subjective ratings using 64-channel EEG. Healthy participants were engaged in self-regulation to increase or decrease pain sensations of the two levels of painful electric stimulation. As expected, both intensity and self-regulation strongly influenced reported pain, decreasing and increasing perceived pain intensity and unpleasantness for down- and up-regulation, respectively. Main effect of pain intensity was expressed in the modulation of N2 and P2 components. In frontal regions we found a non-specific modulation of N1 and P2 potentials by both self-regulation conditions as compared to a control condition, in which participants were asked to experience pain naturally, without influencing it. This suggests that cognitive reappraisal of pain may act through a more general mechanism of mental engagement or cognitive load.

Brain Activity Patterns while Watching Movies Differentiated in Terms of Arousal - New Collection of Dynamic Stimuli and fMRI Study Results
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In recent research, the dynamic stimuli (movies) are used in a variety of experiments carried out not only at the behavioral level, but also in studies implementing advanced neuroimaging methods. Here we present a new standardized scientific tool – Arousing Movie Database - that would aim at explaining which basic emotions are evoked by differentiation of stimuli in terms of arousal value. These selected arousing stimuli were not burdened with the effect of acquaintance. The movies have been divided into three categories: high arousing movies (high arousing extreme sports), low arousing movies (low arousing extreme sports) and neutral movies. The database has been used in fMRI experiment which was carried out in order to observe differences in brain activity patterns during viewing stimuli differentiated in terms of arousal value. 60 healthy right-handed women (aged of 20 – 35 years) participated in the fMRI study. The main effects of movie categories were observed. All results were corrected to family-wise error (FWE) at cluster level (p<.05). Our results revealed significant differences between all movie categories in regions associated with visual and motion processing: primary visual cortex, visual temporal area, lingual gyrus, cuneus. High arousing stimuli also produced activations associated with emotion regulation processing: middle frontal gyrus, posterior and anterior cingulate cortex. Moreover, high arousing movies in comparison to low and neutral movies reveal increased activation in inferior and superior parietal lobule, Extrastriate Body Area (EBA) and Fusiform Body Area (FBA), likely in response to visual body perception, motion and action perception and emotional arousal caused by dynamic body motion presented in films depicting high-risk sports.

Exploring the role of the right temporo-parietal junction in social cognition using transcranial direct current stimulation
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Our ability to control how we represent ourselves or others (e.g. to privilege representations of the self over representations of others, or vice versa) is argued to play a key role for higher order socio-cognitive abilities. At the neural level, controlling self-other representations has been linked to the right temporo-parietal junction (rTPJ), a brain regions thought to contribute to the control of self-other representations. Prior work using transcranial direct current stimulation (tDCCS) has demonstrated that rTPJ plays a key role in imitation inhibition and visual perspective taking, but not Theory of Mind or Self-Referential processing. That is to say that tDCCS to rTPJ
modulated performance on imitation-inhibition and visual perspective taking tasks. While helpful in characterising the role of the rTPJ in self-other control, whether rTPJ plays a key role in domain-general cognitive control (i.e. if tDCS to rTPJ effects non-social cognitive control tasks) or just self-other control remains to be established. Here we conducted an experiment to examine whether tDCS to rTPJ would modulate performance on visual perspective taking (as per previous studies), imitation inhibition tasks (as per previous studies), and domain-general inhibitory control (stop-signal delay task). A within-subject design was employed, with each participant undergoing both active rTPJ and sham rTPJ stimulation. Results showed no significant effect of rTPJ tDCS on imitation inhibition, inhibitory control tasks or visual perspective taking abilities. These data conflict with prior reports of performance change in self-other control following tDCS to the rTPJ. One possibility for this discrepancy relates to differences in experimental design: we employed a within participants design, whereas all prior reports of performance change in self-other control following tDCS to the rTPJ have employed between participant designs. Given practice effects on cognitive control tasks this may mask performance changes following tDCS to rTPJ. Consideration of these methodological issues and the potential of using other types of non-invasive brain stimulation to modulate sub-divisions of the rTPJ with greater precision will be important for future studies.
EP1. Non-parametric approach for analysis of event related activity

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In this study we analysed the influence of reading emotional words on the perception of a subsequent abstract images (in form of QR codes). Especially we were interested in checking the prediction that correlates of brain’s response to stimuli can be manifested in EEG signal in two different ways: phase-locked and non-phase-locked activity. In classical approach, by averaging EEG signals, the phase-locked activity is clearly visible as event-related potential (ERP). On the contrary, a measure of event-related brain dynamics induced by, but not phase-locked to, the onset of the stimuli is canceled out from the average. The analysis of time-frequency distributions of signal energy allows exploring fully non-phase-locked stimulus-induced oscillations. A specific problem arising in analysis of an image perception data are the artifacts caused by the saccadic eye-movement. Independent component analysis (ICA) was used to remove these, and also other types of artifacts like eye blinks, and muscle noise from the signal. A combination of ICA preprocessing and time-frequency analysis revealed decrease activity in beta band and increased activity in gamma band during the perception of the QR-codes.

EP2. Evaluation of Canolty’s methods for assessment of phase to amplitude cross-frequency coupling

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Recent studies indicate that coupling between low- and high-frequency brain rhythms provides valuable information on cognitive processing in humans. The purpose of this study was to investigate efficacy of two methods (proposed by Canolty 2006) that are used to measure the strength of the interaction (phase-to-amplitude modulation) across frequencies. The first method is the modulation index, which is based on analysis of a special signal composed of instantaneous amplitudes of the higher frequency rhythm and instantaneous phase of the lower frequency one. The other method is based on aligning of the normalized time-frequency maps of activity to the phases of a selected frequency band. Both methods were tested on simulated signal and on hippocampal recordings during a short term memory task. We found that the properties of simulated signal, such as signal to noise ratio and ratio of gamma to theta amplitude, may influence the results of the analysis. We determined the range of parameters where both methods give reliable results. The results obtained for hippocampal signal were mostly consistent. Moreover the time-frequency method has a potential to indicate causal relations between the investigated rhythms.
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In light of the reproducibility crisis in science and growing neuro-startup market we believe open science practices are necessary to prevent inflation of p-hacked research and neuro-bubble. One of most successful Polish startup companies, Neuroon, made a claim they can automatically classify sleep stages and increase sleep quality using a small wearable mask. We compared the sleep stage classification produced by Neuroon with a clinical standard polysomnography recording. With this case study we wish to present the tools for open science practices to the neuro and cognitive science community. The original data as well as results of our investigation are available in a public repository.

Sleep consist of several stages, which differ in their function and physiological correlates. The study of sleep using multiple sensors (i.e. EEG, EOG, accelerometer), known as polysomnography, can be employed to classify sleep stages by a specialist following well established guidelines. We have divided the Neuroon validation into three stages. First we assessed whether the EEG signal collected with Neuroon is similar at any point in time to the polysomnography EEG signal. Second we assessed accuracy with which Neuroon predicted the sleep stage. Third we assessed whether there is enough information in the EEG signal to discriminate on that basis between the sleep stages.

EP4. Moving window approach in kCSD method
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One of the technological developments we can observe in the neurosciences is the increasing number of contacts in modern multielectrode arrays which nowadays can offer simultaneous recordings from thousands of contacts. Low-frequency part of the extracellular potential, called the Local Field Potential (LFP), is a useful measure of neural systems activity. Despite its common name LFP is not a local measure – each electrode may record activity observed millimeters away from the source. If only possible it is convenient to estimate the current source density (CSD), the volume density of net transmembrane currents, generating the measured signals. One of the methods which deals with this problem is the kernel Current Source Density (kCSD) method. The kCSD estimates the sources in a family of allowed CSD distributions of dimensionality larger than the number of measurements. The cross-validation technique is used to find the best solution. For high density microelectrode arrays this approach is computationally expensive due to the necessary multiple inversions of huge matrices. What is more, global kCSD reconstruction fails for high spatial frequencies since global regularization smooths out the fine details. In this study we examine a new way of kCSD reconstruction based on moving window approach which may recover more information from the same set of data than standard kCSD method. Moving window approach estimates kCSD locally step by step for every point from estimation space consulting potentials extension. This method may help to increase both quality of reconstruction and speed of calculations for large sets of electrodes.
**EP5. Choosing the best oscillatory power asymmetry metric - results from simulations**
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In many studies, especially on depression and anxiety disorders, asymmetry in power of given oscillatory band (often alpha) is a very often used measure. There are at least three asymmetry metrics used in the literature but guidelines as to which should be preferred in what cases are lacking. The lack of such guidelines is especially relevant given the fact that single-trial power values do not come from a normal but gamma distribution, which is often ignored. We present results of simulations comparing most popular measures of asymmetry. All of these simulations point to simple difference being the best measure (having lowest probability of type II error). The advantage of difference is most striking for single-trial designs, mostly due to the fact that difference of gamma distributions resembles t test distribution (and single-trial approaches are most affected by the fact that the original distribution is gamma-shaped). Additionally we show how to apply each of these measures to EEG data without restricting oneself to only contrasting averages of frequency ranges on pre-selected electrode pairs, but using massive univariate tests with nonparametric cluster-based correction for multiple comparisons.

**EP6. Changes in current sources and connectivity across cortical layers during slow waves in a Fragile X model mouse.**
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Fragile X Syndrome is a commonly inherited form of intellectual disability and one of the leading genetic causes for autism spectrum disorder. The Fmr1 knock-out (KO) mouse is a valuable animal model to study the effect of functional loss of the Fragile X mental retardation protein (FMRP). We focused our study on the characteristics of the slow-waves oscillations during anesthesia in the neocortical network of wild type (WT) and Fmr1 KO mice model. This spontaneous thalamocortical activity can propagate as UP and DOWN states that are related to the collective switch of the neuronal membrane potential between the depolarized and hyperpolarized states, respectively. Slow waves have previously been studied during slow-wave sleep or quiet wakefulness and were characterized on the basis of the local field potential (LFP) in vivo and in vitro recordings. Using the multiunit activity (MUA) analysis for the UP and DOWN state detection, we compared UP states duration in sick and healthy mice across the cortical column. To better characterize this spontaneous network activity we also tried a new analytic approach – kernel Current Source Density (kCSD) method – to estimate the changes in current sources during slow oscillations. Moreover, previous histology studies reported presence of long thin dendrite branches in Fmr1 KO mice brains [1]. We think that this may contribute to the information transfer inside cortical column. To evaluate changes in connectivity across different cortical layers direct transfer function (DTF) analysis has been adapted to the multichannel signal. Our preliminary results suggest that there are some differences in information propagation across cortical columns in fragile X syndrome model mice.
EP7. Does isolated stimulus probability have an effect on Contingent Negative Variation?
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The response preparation is a basic brain function, which is commonly studied with event-related potential parameter called Contingent Negative Variation (CNV). One of the fundamental CNV properties is a sensitivity of this parameter to the probability information provided by precue. The higher probability information results in the higher CNV amplitude. However, since both stimulus and response probabilities were confounded and equal in previous studies, it is still unknown, which exact probability has an effect on the CNV amplitude. In order to study this query we used two different tasks: the simple and the compound one. Differently than the simple task, the compound task had not only simple response, but also a convergent one (assigned to the two different stimuli) – the standard method to split up the stimulus and response probabilities. Therefore, despite the equal response probability in the both tasks (66.6%) the stimulus probability was the 66.6% only in the simple task, while in the compound task it was 33.3%. The results of our study revealed no difference of CNV amplitude between the tasks with different stimulus probability information. Therefore, it was found that isolated stimulus probability had no effect on the CNV. This finding suggests concluding that the CNV is sensitive only to the response probability. Additionally the experimental design of our study allowed solving methodological problem in the investigation of another query: if the convergent response is slower than a simple one. The results revealed not only the lower speed of convergent response, but also its lower stability, assessed with intra-individual reaction time variability.
The possible diet is a crucial to maintain the right function of the nervous system. Over several decades can observe abuse of fat in daily nutrition. This tendency can lead to very serious health consequences. In 2014, more than 1.9 billion adults, were overweight. Of these over 600 million were obese. This diet can lead to metabolic syndrome, diabetes and stroke. Besides, it can have influences on central nervous system. Demonstrated that excessive consumption of high fat diet (HFD) can contribute to loss of cognitive function, increased risk of Alzheimer's disease and speeding up aging process. So, in a world where population of aging and obese people is so much higher, which seems to be necessary finding the the mechanism of brain's dysfunction connected with excessive consumption of HFD. Definitely less studied and less well known is the mechanism of the impact of diet on the functioning of microglial cells / astrocytes. The latest experiments shows that excessive consumption of HFD have influence on neuroinflammatory markers which labels activated microglial cells. Furthermore, results of research demonstrate that in rodents high-fat feeding caused astrocyte activation, known as reactive astrogliosis and noticeable morphological changes in population of these cells. During the presentation will be presented the recent studies shows functional and morphological changes of glial cells. Moreover will be demonstrated our preliminary results connected with this subject. We used to this experiments male Wistar rats which were fed for 6 months high fat diet (70% energy from fat). After this period we tested histological changes in population of glial cells and disturbances in expression proteins in appropriate structures (hippocampus and frontal cortex). Our results shows that HFD can have influence on synthesis very common proteins located in astrocytes- GFAP (glial fibrillary acidic protein) and GS (glutamine synthesis). Higher expression of GFAP and lower expression of GS after the HFD treatment can suggest that this diet can have detrimental effect on functioning of astroglia.

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BACKGROUND/AIMS: Unilateral spatial neglect, or neglect syndrome, is a behavioral syndrome, occurring after brain injury, which leads to inattention to contralesional side of space. It is more commonly observed after cerebral infarction, which occurs on the right side of the brain, and affects up to two thirds of patients with right hemisphere lesions. This condition is more frequently associated with lesions of the inferior parietal lobule or temporoparietal region. As a result, patients are not aware of visual, somatosensory or auditory stimuli on the contralesional side. Neglect syndrome can also be exacerbated by anosognosia, which usually accompany right hemisphere lesions. All above mentioned leads to reduction of rehabilitation potential of such patients and decreases adaptation of this patients in common life after the lesion. Therefore, neglect syndrome must be properly diagnosed and treated. METHODS: We examined 35 patients, who underwent treatment for right-hemisphere stroke in the neurological departments in the period from December 2015 to September 2016. The
average age of the patients was 65.9±12.0. To solve the problems, we used double-simultaneous stimulation for extinction in the visual, auditory, somatosensory, or motor modalities and battery of neuropsychological tests, which include line crossing, letter cancellation (in rows and randomly positioned), figure copying (a four-pointed star, a Necker cube and a daisy), representational drawing (clock face, face of the human and a butterfly), map navigation, line bisection and picture scanning.

RESULTS: For 13 of 35 patients (37%) was diagnosed neglect syndrome. Among sensory tests most informative appeared double visual stimulation (76.9%). Figure copying, map navigation, picture scanning helped to reveal neglect syndrome in 84.6%, 92.3% and 84.6% respectively. The most informative test among figure copying was a daisy (76.9%), among representational drawing – clock test (53.8%). The feature of the patients, diagnosed with neglect syndrome, was that they tended to begin tests from the right side of the paper or even use only right side of the paper.

CONCLUSION: Unilateral spatial neglect is rather widely-spread condition (37% in our research). Most informative test among sensory tests appeared double visual stimulation. Most convenient and clinically significant neuropsychological tests were letters cancellation, figure copying, map navigation, line bisection, picture scanning.

EP10. Effects of acute and repeated administration of tianeptine on binding of [3H]CP55,940 to cannabinoid (CB)1 receptors in rat brain structures
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Depression is one of the most common mental disorders in the 21st century and affects about 121 million people worldwide. Current research assume that depression is caused by a combination of genetic, biological, environmental and psychological factors and efficacy of modern pharmacotherapy is not fully satisfactory. Different mechanisms of action of antidepressant drugs suggest that this drug interaction with the direct target molecule is not responsible for the therapeutic effect but rather neuroadaptive mechanisms have a significance. The potential participation of the endocannabinoid system in the pathogenesis of depression and in the mechanism of action of antidepressants has been highlighted in recent years. The aim of this study was to investigate the effect of the clinically effective atypical antidepressant - tianeptine (TIA, 10 mg/kg) on the cannabinoid (CB)1 receptor labelling pattern in selected rat brain structures. Male Wistar rats received TIA intraperitoneally acutely and chronically for 14 days. Twenty four hours after the last drug administration the animals were decapitated and their brain CB1 receptors were analyzed using quantitative autoradiography with the CB1 receptor agonist – [3H]CP55,940. Acute and repeated administration of TIA increased the levels of [3H]CP55,940 binding in the motor cortex (IV-VI layers), frontal cortex (V layer) and hippocampus areas, including, CA1 layer (bregma -3.30) and CA3 layer (bregma -3.30). Significant increases in [3H]CP55,940 binding were also seen in the infralimbic cortex, cingulate cortex and CA1 layer (hippocampus bregma -5.20), after chronic TIA administration. Our data indicate the potential engagement of CB1 receptors in the effects of TIA, however further investigation is required to assess the underlying mechanism of such interaction. This study was supported by the research grant UMO-2012/05/B/NZ7/02589 from the National Science Centre and by the statutory funds of the Institute of Pharmacology (Kraków, Poland).
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Malformations of cortical development are a broad range of central nervous system anomalies heavily implicated in the pathogenesis of intractable epilepsy, especially in children. Focal cortical dysplasia (FCD) is both the most common, and the most complex of these diseases, with three different types (and many subtypes) described based on anatomical and cytological pathologies. Multiple animal models were developed over the years to recapitulate FCD-like pathologies in an experimental setting. Among them, prenatal gamma irradiation in rats is a well-described model presenting characteristics similar to FCD type 1 in humans, such as disturbed cortical lamination and organization, subcortical heterotopias, hippocampal pyramidal layer dispersion and a number of cellular pathologies. These pathologies include interneuron loss and alterations in astrocyte function and density. Different time points of irradiation, corresponding to specific developmental milestones, generate distinct patterns of dysplasia. Early irradiation (gestation days E13-E15) results in greater changes in overall brain volume and massive heterotopias, while later (E19) may be associated with astrocytic deficits. Here, we assayed astrocytic protein expression in hippocampi obtained from adult rats with cerebral dysplasia generated at prenatal days E13, E15, E17 or E19 as well as non-irradiated controls. Male Wistar rats were sacrificed at P80, the hippocampi were bilaterally dissected and homogenized. These samples were then used for immunoblotting with antibodies against glial fibrillary acidic protein (GFAP), connexin 43 (Cx43), inwardly rectifying potassium channel Kir4.1 and glutamate receptor subunit Glur4. Chemiluminescent Western signal was normalized to total protein load per lane obtained from Stain-Free (UV-catalyzed trihalo reaction) signal. Between-group normalized protein levels were compared with analysis of variance (ANOVA). While GFAP immunoreactivity is unchanged in all experimental groups, the expression levels of Cx43, Glur4 and Kir4.1 are decreased in the late-irradiated E19 group, in contrast with E13-E17. Since these proteins may be involved in processes related to epilepsy, we posit the time-dependent decreases in their tissue levels may reflect functional changes in astroglial involvement in epileptogenesis. This work was supported by the National Centre for Science grant DEC-2012/05/N/NZ2/00641.

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Background and aim: Dysregulation of the ceramide metabolism (e.g., by the acid sphingomyelinase) has been proposed as an important factor in the pathogenesis of depressive disorders. Moreover, some antidepressant drugs function as the acid sphingomyelinase inhibitors and decrease ceramide levels in the rat hippocampus. However, the role of the path ceramide synthesis de novo is unknown. The aim of this study was to analyse effects of antidepressants having different mechanisms of actions and chemical structures (imipramine, tianeptine, escitalopram) on several ceramide synthases in the rat hippocampus, cerebellum and striatum. Materials and methods: Male Wistar rats received imipramine (IMI, 15 mg/kg), tianeptine (TIA, 10 mg/kg), escitalopram (ESC, 10 mg/kg) or
corresponding vehicles acutely or chronically (for 14 days). Twenty four hours after the last injection the animals were decapitated. Brain structures were analyzed using Western Blot.

Results: We found significant increases in the synthase ceramide 2 levels after acute administration of IMI or TIA in the hippocampus as well as after chronic administration of IMI in the cerebellum. Acute administration of TIA or ESC observed significant increase in the synthase ceramide 2 levels in the striatum. Acute and chronic administration of IMI resulted in a significant increase in the level of the ceramide synthase 4 in the cerebellum and hippocampus, respectively. In turn acute administration of IMI causes significant decrease in the level of the ceramide synthase 4 in the striatum. On the other hand, an acute administration of TIA induced a significant decrease in the synthase ceramide 4 protein expression in the hippocampus. After chronic administration of ESC a significant increase in the synthase ceramide 5 levels in the hippocampus and striatum was found; the increase in the latter enzyme was noted for the chronic administration of IMI in the hippocampus.

Conclusions: Our findings indicate that different antidepressant drugs alter the expression of number of ceramide synthesis what may further highlight the role of this sphingolipid in the pathophysiology of depression.

EP13. Slower Baseline Respiration Rate Is Associated with More Accurate Heartbeat Detection
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Interest and progress in studying interoceptive facets has been rising for the past quarter of century. Schandry’s heartbeat counting task (HCT) is a most pervasive method to investigate individual differences in interoceptive accuracy (IAc). Although there were suggestions that slowing the breathing rate (BR) or holding breath may potentially improve perception of heartbeats, these suggestions lacked empirical investigations. To elucidate this question, in current study, association between IAc and ECG-derived BR was examined in sample of 35 healthy young participants (18 males and 17 females, mean age 24.0 years). Slower baseline respiration was associated with more accurate heartbeat detection during subsequent HCT in the total sample (Spearman rho = -0.357, p = 0.036), however after investigating those relations in males and females separately, the association was observed only in females (Spearman rho = -0.591, p = 0.013) and not in males. Besides, in the subsample of participants who slowed down their respiration from baseline to HCT (8 females and 7 males), IAc was also related with slower BR during baseline (Spearman rho = -0.693, p = 0.006) and during HCT (Spearman rho = -0.800, p = 0.0006). In conclusion, the results partially confirms that respiration may influence cardioceptive accuracy.

EP14. Rat model of femoral to sciatic nerve transfer
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Background: Complete peripheral nerve injury results in paralysis. Nerve transfer (neurotization) is a reconstructive procedure including repair of distal denervated nerve (recipient) by connecting it with proximal, healthy nerve with less significant function (donor). The aim of this study was...
to determine whether femoral (donor) to sciatic (recipient) nerve neurotization in rat model is possible to perform with restoration of sciatic nerve function, which has never been described in literature. Methods: 3 SPRD male rats (8-10 weeks old) were treated with left side femoral to sciatic nerve neurotizations on the level of lumbar spine and observed for 50 days. On day 50 retrograde fluorescent axonal tracers (1μL, 2% solution) were injected into sciatic nerve on the level of proximal thigh (True Blue into left nerve and Diamidino Yellow into right nerve). On day 57 pinch test of each hind limb was performed to evaluate restoration of sensory and motor function, afterwards rats were anesthetized and sacrificed. Spinal cord and sciatic nerves were harvested and analyzed histologically. Results: During pinch test, rats were retracting hind limbs after needle pinching, left limb retraction compared to right limb was: less intense and observed after stronger stimulus. Tracers' injections resulted in retrograde axonal transport of fluorescent tracers from site of injection to the body of neural cell (motoneuron) located in the spinal cord. Tracers were visualized with fluorescent and confocal microscopy of spinal cord sections. True Blue in cells on left side and higher segment of spinal cord than Diamidino Yellow present in cells on right side and lower segment of spinal cord. Conclusion: On the side of performed neurotization (left side) innervation originated from higher segments of spinal cord than on the untreated (right) side, which implies that femoral nerve axons grew into damaged sciatic nerve and reinnervated it's distal targets with good functional outcome, which was confirmed by pinch test (restoration of sensory and motor function).

EP15. Resting state brain activity differences among schizophrenic patients with suicidal ideation
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Objective: To determine neural basis of suicidal thoughts among patients with schizophrenia using resting state brain activity approach. Method: The study involved group of patients with suicidal thoughts (N=5), obtained by the Beck Depression Inventory (BDI-I), and group of patients without suicidal thoughts (N=5). Level of schizophrenia symptoms was controlled in both groups. 8 min long resting state brain activity was acquired using functional magnetic resonance imaging technique. After data preprocessing, regional homogeneity analysis (ReHo) was performed, in order to provide information about presence, and the nature of the differences in neuronal activity associated with suicidal thoughts. ReHo activity maps were then compared between patients with and without suicidal thoughts using statistical testing methods. Results: In accordance with the hypothesis, regional homogeneity analyses provide information about brain regions which significantly differentiate both groups. Patients with suicide thoughts, compared to patients without suicidal thoughts, were characterized by significant (cluster-level p = 0.02 FWE corrected, k=12), increased regional homogeneity in Left Middle Temporal Gyrus. After controlling for depression severity, added as a covariant to the analysis, patients with suicide thoughts were characterized by one significant (cluster level p< 0.01, k=5), brain region with increased regional homogeneity: Right Superior Temporal Gyrus. Discussion: The results reflect the functional change of brain related to suicidal thoughts among patients with schizophrenia. Increased regional homogeneity in Right Superior Temporal Gyrus turns out to be independent of depression severity, which is one of the most important factors related to suicide.
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Our current understanding of sequence of molecular events leading to early-onset Alzheimer’s disease is limited due to a lack of appropriate in vitro model. Therefore, a generation of patient-derived induced pluripotent stem (iPS) cells, further converted into neuroepithelial-like stem (NES) cells, and differentiated into neurons can offer an alternative approach. The aim of this study was differentiation of NES cells into nerve cells. For this purpose primary cell lines of skin fibroblasts derived from a patient with Alzheimer’s disease carrying R307S mutation in PSEN1 were reprogrammed into iPS using Yamanaka factors. Subsequently iPS cells were subjected to neural induction into NES cells. Established NES cell lines were cultured and differentiated into neurons, which were validated by immunocytochemical staining with neuronal markers and glial marker. In order to characterize the obtained neuronal subtypes, mRNA expression profile of different neural lineages was analyzed. The latter analysis revealed that this differentiation protocol gave rise to neurons of predominantly GABAergic phenotype with some glia. We also compared NES with fibroblasts in terms of the expression and localization of BRCA1 and H2AX. Summarizing, this study showed derivation steps of neurons from Alzheimer’s patient and provided material for future studies on AD pathomechanism using “disease-in-a-dish” approach.

EP17. The effect of benzophenone derivatives on the viability of neuroblastoma cell line (SH-SY5Y)
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Ultraviolet filters (UV filters) are widely used for protection against sunburns and in order to reduce the risk of skin cancer in sunscreens and in variety of cosmetic products or materials. However, some UV filters can be absorbed through skin and by consuming contaminated food. UV filters are added to consumer sunscreen products in a concentration of up to 10%, but remain essentially unidentified in technical products. Exposure of human body is significant and still growing. Most common group of organic UV filters are benzophenone derivatives such as benzophenone-2 (BP-2) and benzophenone-3 (BP-3) which are absorbed through skin to the greatest extent and exert systemic effects. These compounds as highly lipophilic are likely to pass through the blood-brain barrier and, therefore, their toxic effects on the central nervous system cells cannot be ruled out. In the present study, we investigated the effect of benzophenone derivatives on cell viability and caspase-3 activity in SH-SY5Y cells. It has been found that BP-2 and BP-3 present in the culture medium for 72 h in high concentration (10−5 M) produced a significant cytotoxic effect, as determined both by the MTT reduction test and LDH release assay. In contrast to necrotic changes benzophenone derivatives
increased caspase-3 activity in much lower concentrations (from 10−8 to 10−7 M). Proapoptotic properties of the test compounds were positively verified by Hoechst staining. The obtained results indicated that benzophenone derivatives adversely affected the viability of neuroblastoma cells, most likely by enhancing the process of apoptosis. Since human exposure to UV filters is significant and still growing these compound should be taken into consideration as one of the possible factors involved in pathogenesis of neurodegenerative diseases. However, such conclusion must be confirmed in in vivo studies.

**EP18. The influence of nanoparticles and microparticles of red phosphorus and single-wall carbon nanotubes on spheroid culture of glioblastoma line U87**

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Glioblastoma is the most common and malignant brain tumor in adults. Recently, the identification of anti-cancer drugs is mainly based on the assessment of monolayer cellular cultures and animal models studies. 3D cellular culture model combines high-throughput of in vitro and many features found within tumors in vivo, including cell-cell and cell-matrix interactions. The aim of this study was to evaluate the effect of nanoparticles and microparticles of red phosphorus and single-wall carbon nanotubes on tumor spheroids of glioblastoma line U87. An assay based on measuring the activity of cytosolic enzyme lactate dehydrogenase (LDH) was used to evaluate the cytotoxicity of nanostructures. Cells structure analysis was performed by computer image analysis of fluorescently-labeled phalloidin, which visualize F-actin. The results of the adherent culture were compared to the spherical one. The data suggest that there are inequalities in the results of tests performed on 3D and 2D in vitro model. This research indicates that there are differences in the formation of spheroid's structure under the influence of the various nanostructures.

**EP19. Optimization of murine dental pulp stem cells isolation for neural tissue regeneration**

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Dental pulp stem cells (DPSC) are ectoderm-origin cells residing in pulp chamber of mammalian teeth. Human DPSC are reported to be multipotent stem cells, posses mesenchymal characteristic and secret a numbers of paracrine factors involved in tissue regeneration. Impact of those factors was confirmed by in vivo models for both cardiac and neural regeneration in immunodeficient rodents. However less is known about murine dental pulp stem cells (mDPSC). For better characterization of mDPSC we optimized isolation protocol of murine dental pulp and compare obtained cells with murine BM-MSC. Dental pulp stem cells and bone marrow stromal cells were isolated from 10 C57BL/6 mice. Isolation of BM-MSC was conducted by standard protocol used for that issue. For mDPSC isolation we compared three isolation methods: flashing, explant method and crushing. Adherent cells were cultured under standard condition and after 21 days qRT-PCR were conducted. Two of mentioned protocols of dental pulp isolation resulted in successful isolation of adherent dental pulp cells – flashing and crushing. Cells exhibit mesenchymal morphology with prominent
nucleus and were rapidly proliferating. Genetic analysis exhibited differences in expression of all investigated genes. Nestin expression was higher for mDPSC in comparison to BM-MSC. High expression of nestin is characteristic for neural stem and progenitor cells and was reported in studies about hDPSC. Murine DPSC exhibited lower expression of pluripotency genes (Oct4, Nanog) and Eras in comparison to BM-MSC. Those results could be connected with fact that mDPSC start to differentiate or heterogeneity of obtained cells. Further investigation about heterogeneity of obtained adherent mDPSC culture are needed to better explain those genetic analysis results.

EP20. One week stimulation of low-threshold proprioceptive fibers increases density of glutamatergic and cholinergic boutons on the ankle extensor α-motoneurons in the adult rat

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Chronic stimulation of low-threshold muscle afferents (Ia) in the tibial nerve in awake rats causes an increase of neurotrophin NT3 in the spinal cord and soleus muscle, being a good predictor of synaptic plasticity in this spinal circuit (Gajewska-Woźniak et al., 2013). We hypothesized that this stimulation can reinforce NT3-dependent, glutamatergic Ia input to the extensor α-motoneurons (MNs) but also affect indirectly, through spinal interneurons, cholinergic neurons of V0c group modulating activity of MNs via C-terminals. To verify it, we examined the effectiveness of the same paradigm of stimulation on morphology and density of both inputs to MNs of the lateral gastrocnemius (LG) muscle. Tibial nerve was stimulated unilaterally for 7 days with continuous bursts of 3 pulses in four 20 min sessions daily. The Hoffmann reflex recorded from the soleus muscle, LG synergist, allowed controlling low-threshold stimulation. LG MNs were identified with intramuscularly injected tracer. Glutamatergic Ia terminals and cholinergic C-terminals were detected immunohistochemically, using specific anti-VGLUT1 and anti-VAChT antibodies. Quantitative analysis of terminals on LG MNs, which were captured with confocal microscopy, revealed that the number of VGLUT1 and of VChAT immunoreactive terminals contacting the soma increased after stimulation by 35% and by 25%, respectively, comparing to the sham-stimulated side. Also their aggregate volume was increased. Analysis of frequency distribution of boutons within arbitrarily distinguished 4 classes of terminals showed that enrichment occurred in groups of the smallest boutons (VGLUT1: 1-5 μm³, VChAT: 5-10 μm³) in expense of the larger boutons (volume > 10 μm³). This set of data may indicate that 7 days of stimulation of Ia afferents is sufficient to cause a formation of new glutamatergic and cholinergic terminals, both characterized by their small volume at this stage. In the next step we examined the effect of this stimulation in the rats with complete spinal cord transection. We found that it was not sufficient to compensate for the deficit of glutamatergic innervation, which occurred 1 week after spinalization. To conclude, 1 week of stimulation of proprioceptive Ia input to LG MNs is sufficient to enrich their direct glutamatergic but also indirect cholinergic innervation in non-spinal, but not in the spinal rats. A study investigating the effect of longer stimulation is in progress.

Support: NCN grant 2013/09/B/NZ4/03306

EP21. NMDA receptors as promising target for pharmacotherapy in severe mental disorders

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Severe mental disorders, including bipolar affective disorder (BPAD), schizophrenia (SZ) and major depressive disorder (MDD), despite extensive research, remain significantly challenging for modern psychoneuropharmacology. I will briefly highlight most troublesome aspects of these disorders
and the relevance of glutaminergic system in their neurophysiology, along with the promising effects of experimental treatments with pharmacological agents that possess NMDA antagonist properties. Underlying causes, neurochemical mechanisms and genetic factors of aforementioned mental disorders aren’t fully known, and existing knowledge, including knowledge that underlies the development of pharmaceuticals targetting these disorders, is often of a speculative, sometimes controversial nature. It is worth to mention that existing treatments are often linked to troublesome – in some cases potentially fatal – side effects, and their efficacy, in many cases, is not fully satisfying, as shown in publications focused on the quality of life in pharmacologically treated people suffering from mental disorders. Therefore, there still is a need to consider and conduct the research of different approaches to these mental disorders, including, if possible, taking preventive measures, along with consideration of new neuropharmacological targets for pharmacotherapy. There is a growing body of evidence that NMDA receptors play an important role in neurophysiology of many mental disorders, including BPAD, SZ and MDD. Pharmacological agents with NMDA antagonist properties – dextromethorphan, ketamine and memantine, show promising results in trials as adjuvant and/or monotherapy agents in the treatment of aforementioned disorders, both in their efficacy and tolerance, although randomized, controlled clinical trials including higher numbers of people would be desired to confirm their value.
EP22. Optogenetics at electrophysiologist’s site – neuronal stimulation by using light during in vitro patch clamp experiments

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Classical method used to activate neurons is based on electrical stimulation. This method is effective, however has some limitation. The main drawback is the lack of possibility to selectively stimulate specific groups of neurons. One option to cope with this difficulty is optogenetics - new method based on stimulation of genetically modified cells by using light. Genetic engineering introduces light sensitive channels into membranes of specific populations of neurons. Light impulses delivered with high temporal resolution open the channels and depolarize the cells. We tested the characteristic of optogenetical stimulation of cortical cells by means of whole-cell patch-clamp in-vitro recordings.

In order to introduce channelrhodopsin (ChR2) into neurones we injected young rats with a viral vector AAV-hSyn-ChR2-EYFP bearing ChR2 and fluorescent protein genes under synapsin promoter. After 2-3 weeks necessary for effective gene expression the rats were sacrificed, their brains extracted cut into 380 μm slice. During experiments, we first checked the neuronal responses to direct current injection to the cell through the recording pipette (-100 - 100 pA). Next we stimulated cells with a blue light (470 nm), which opens channelrhodopsin. We tested the responses to various frequencies of stimulation (20 – 200 Hz) and various durations of light impulses (2 -20 ms). Our results show, that extending light pulse duration or increasing the frequency of stimulation gives the higher depolarization and (in case of exceeding action potential threshold) higher probability of evoking an action potential. Moreover, we can conclude that optical stimulation can reproduce the electrophysiological effect of direct current injections and but gives more possibilities to study the neural network also in vivo. [Supported by Polish National Science Centre grant 2013/08/W/NZ4/00691].

EP23. Action potential threshold of spinal motoneurons during scratching and swimming

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Motoneurons integrate excitatory and inhibitory synaptic inputs and when the membrane potential of a motoneuron is depolarized sufficiently to exceed the threshold potential, an action potential is generated. The threshold for action potential generation can be dynamic and it provides a mechanism for the modulation of the excitability of a neuron. During functional spinal neural network activity motoneurons receive intense synaptic input, and this could modulate the threshold potential. In the present study we investigated how the threshold potential is affected during fictive scratching and fictive swimming. We performed intracellular electrical recordings from turtle spinal motoneurons in ex vivo carapace–spinal cord preparation and in spinal cord slices. Fictive scratching was induced by mechanical stimulation of carapace. Fictive swimming was induced by electrical stimulation of the descending cDLF tract. We found that the threshold potential depolarizes in bursts of action potentials generated during scratching and swimming. The magnitude of depolarization of the threshold during functional spinal neural network activity is similar to the one during stimulation of a motoneuron with a rectangular current pulse. Comparison of action potential threshold before and after functional spinal neural network activity show that synaptic inputs does not affect the threshold
of the first action potential in firing bursts during scratching and swimming.

**EP24. Alcohol differently affects processing of visual stimulus onset and offset**

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Responses to stimulus onset and offset are important in coding stimulus duration. Previous studies have shown that ON and OFF responses may vary between sensory systems and stimulus processing phases. Effect of ethanol on the CNS is typically manifested as altered visual perception, which is caused by incorrect processing of sensory information. Effect of acute ethanol on the flash VEPs was investigated in numerous studies. However, there are many unanswered questions, particularly related to the effect of alcohol on visual system ON and OFF responses. The aim of the present study was to investigate effect of alcohol on the rat visual cortex visually evoked potentials to visual stimulus onset and offset. We investigated effects of acute alcohol administration on visually evoked potentials (VEPs) in anesthetized adult rats (n=10). The effect of alcohol on VEPs latency was observed for one hour after intraperitoneal injections of ethanol. We used two ethanol doses - 1g/kg and 2g/kg. Three VEPs components - N53, P98 and N145 – elicited after stimulus onset and offset were analyzed. Alcohol increases latency (except for component N144 from OFF responses) and reduces amplitudes (except for component N144 from ON and OFF responses) of all components in ON and OFF responses. Independent of dose, alcohol differently affects components of ON and OFF responses: increases latency differences of N144 component. Our results show that activation of visual system during ON response to 500 ms visual stimulus is qualitatively different from that during OFF response. Both ethanol doses increased latency of all three VEP components during an hour and differentially affect ON and OFF responses, in particular leading to changes in perception of stimulus duration.

**EP25. Activation of oxytocin receptor contributes to the neuronal growth**

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Objectives. Neuropeptide oxytocin is abundantly produced in the brain. Oxytocin receptors have been demonstrated in several areas of the brain, and they were found in cultured neuroblastoma cells as well. Oxytocin increases growth and viability of neuronal cells, however direct involvement of oxytocin receptor is not known. Therefore, the aim of the present study was two fold 1) to determine the effect of oxytocin and oxytocin receptor antagonists on neuronal proliferation and 2) to measure the effect of oxytocin on neuronal differentiation. Methods. Experiment I. Proliferation of human SH-SY5Y cells was tested in response to incubation with oxytocin, oxytocin receptor antagonists atosiban and L-371,257 after 24–96 h. Cell viability, which is closely related to proliferation was evaluated by the neutral red method of monitoring in vitro cytotoxicity. Experiment II. Neuronal differentiation was evaluated by observing neurite length and number. Neurite outgrowth was measured in response to 48h incubation with oxytocin, L-371,257 and their combination in primary
cortico-hippocampal neurons isolated from newborn mice. All-trans retinoic acid (ATRA) was used as a positive control. Neurite number was evaluated after 12h incubation of SH-SY5Y cells with oxytocin and L-371,257. Results. Oxytocin increased cell number while both antagonists decreased number of cells compared to control. Oxytocin increased viability of cells in test of cytotoxicity after 48h. Neurite length of primary neurons increased in response to both ATRA and oxytocin; however L-371,257 co-treatment significantly antagonized oxytocin’s effect with no effect on cell growth in ATRA treated cells. Oxytocin increased neurite number in SH-SY5Y cells in comparison to control group. Conclusion. The results suggest that oxytocin receptors play a role in neuronal proliferation and differentiation. Moreover, oxytocin receptors are involved in neurite outgrowth at least in certain types of neuronal cells. Acknowledgement. Supported by grants APVV-15-0205 and 2/0119/15.

EP26. Oxytocin modulates response of SH-SY5Y cells to inflammatory stimuli
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Oxytocin, released in response to different physiological stimuli, plays a role in neuronal differentiation and growth. It has been suggested that oxytocin has protective effects against inflammation and consequences of oxidative stress. The mechanisms how oxytocin could be involved in neuronal protection is unclear. Therefore, the aim of the present work was to determine the effect of incubation of SH-SY5Y cells with 10 μg/ml lipopolysaccharide (LPS), 1 ng/ml tumor necrosis factor alpha (TNFα) and 50 μM hydrogen peroxide on the viability and morphology of SH-SY5Y cells with/without the presence of 1 μM oxytocin for 48 hours. Next, the expression of different apoptotic proteins was analyzed. Oxytocin significantly increased cell viability and neurite outgrowth of SH-SY5Y cells. A decrease of length of neurites observed under influence of LPS and TNFα was partially compensated by oxytocin. LPS and peroxide resulted in a decrease of cell viability. Hydrogen peroxide induced significant elevation of pro-apoptotic proteins with the significant effect on BAX concentration, this effect was partially blocked by oxytocin treatment. The results suggest that oxytocin is involved in the regulation of cell death and the activation of oxytocin receptors may prevent deterioration of neuronal cells in response to environmental factors. Supported by grants APVV-15-0205, 2/0116/16 and 2/0119/15.

EP27. Systemic inflammation at different developmental stages increases numbers of nNOS-immunopositive neurons in response to status epilepticus evoked in adulthood
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Objectives Neuronal nitric oxide synthase (nNOS) is the neuronal form of enzyme responsible for the synthesis of nitric oxide - signaling molecule crucial for many physiological processes such as synaptic plasticity and inflammation. In the hippocampus nNOS is primarily expressed by subpopulations of GABAergic interneurons important in many physiological processes. Moreover, these interneurons may contribute to pathological states related to dysfunction of NO production and release, e.g. in neuronal death and epilepsy. However the results of dysfunctional nNOS activity differ in distinct
epilepsy models. According to previous studies, neuroinflammation may lead to an increase in seizure susceptibility and trigger epileptogenesis. However, emerging experimental evidence indicates that early age inflammation acting as a preconditioning factor may also have protective effects. The aim of this study was to examine long term effects of systemic inflammation induced at different postnatal developmental stages on the nNOS+ cell population within hippocampal formation in response to status epilepticus evoked in adulthood. Methodology Wistar rats were injected intraperitoneally with LPS on postnatal days 6 (P06) or 30 (P30). When became two-month-old, they were injected with pilocarpine to evoke status epilepticus and sacrificed three days later. Brain sections were then processed for nNOS immunohistochemistry and nNOS+ neurons were counted bilaterally within CA1, CA2/3 and DG regions of the dorsal part of hippocampal formation. Results LPS injections alone on P06 or P30 caused significant increases in numbers of nNOS+ cells but only within the CA1 area when compared to naïve animals. The seizures induced in LPS-untreated animals led to significant decreases of nNOS cell numbers in both CA1 and CA2/3 area. However, in rats injected with LPS on P06 and P30 the effect of seizures was significantly lower than in LPS-untreated controls. Conclusions Transient inflammation induced during the brain development led to permanent increases of nNOS+ neuronal population in the adult brain. The inflammation induced on P06 and P30 could also prevent large seizure-related reduction of the cells. This might result from long-term changes in nervous-tissue reactivity - preconditioning. Supported by the NSC grant:UMO-2012/05/B/NZ4/02406.

EP28. Degradation resistant PSD-95 enlarges dendritic spines
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The ability of the nervous system to learn and form new memories, hence adapt, is believed to be based on activity-dependent modifications of synaptic connections, accompanied by their morphological alterations. It is not yet known what molecular mechanisms underlie these morphological changes. PSD-95 is a major scaffold protein of the postsynaptic density (PSD), where it is highly abundant. PSD-95 dependent protein complexes are known to regulate mechanisms underlying LTP (long-term potentiation). PSD-95 activity-dependent trafficking out of dendritic spines and its degradation are regulated by phosphorylation of serine 73 (S73), the protein phosphorylation site, via CaMKII. In this study we tested the role of overexpression of 3 forms of PSD-95 in altering dendritic spine morphology. First we cloned WPRE (Woodchuck Hepatitis Virus posttranscriptional regulatory element) sequence into AAV vectors, containing psd-95 sequence, to enhance transcription. Plasmids we used contained 3 forms of PSD-95 fused with fluorescent mCherry: wild type PSD-95, non-phosphorylatable PSD-95 S73A and phosphomimetic PSD-95 S73D. mCherry coding vector was used as a control. We then transfected dissociated hippocampal culture with the constructs using lipofectamine and performed immunostaining for PSD-95 and mCherry. The cells were visualized using confocal microscopy. We measured spine density, area and length with Spine Magick! software. Spines in cells overexpressing non-phosphorylatable PSD-95 S73A were significantly bigger and longer than PSD-95wt cells and the phosphomimetic PSD-95 S73D cells. These results confirm the crucial role of PSD-95 and its phosphorylation via CaMKII in spine growth termination. PSD-95 trafficking seems to play a major role in dynamic changes of spine morphology.
EP29. In vitro model for the studies on the biology of rat oligodendrocyte progenitor cells
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Background: Oligodendrocyte progenitor cells (OPCs) constitute the main population of dividing cells in central nervous system. They are mobilized in response to various injuries followed by hypomyelination and demyelination. In these cases OPCs may give rise to mature, myelinating oligodendrocytes, but, also support neuroregeneration. The aim of our study was to settle culture conditions which may allow us to trace the active compounds released by OPCs in response to damaging conditions. Thus, we evaluated growth of rat OPCs in vitro in serum-free medium and physiological normoxia (2-5% O2) compared to standard conditions. Materials and methods: OPCs were obtained from rat primary glial cultures. These were established from brain hemispheres of 2-day-old Wistar rat pups. Mixed glial cultures grew under 21% O2 in DMEM containing 10% FBS. After 12 days, OPCs were isolated with a shaking method which uses differential adherent properties of neural cell types. Oligodendrocyte progenitors were then plated at densities between 1.2 and 6 x 10⁴ cells/cm². Cells were cultured under 5% or 21% O2 either in serum-free medium or supplemented with 1% FBS. After 2DIV or 5DIV the cells were fixed and identified by immunolabeling with antibodies against Ki67 for proliferating cells, NG2 for OPCs, GalC for immature oligodendrocytes, MBP and PLP for myelinating cells. Results: The 1% FBS addition to culture medium enhanced the OPCs differentiation at low oxygen level, but had no influence on the number of GalC- and MBP-positive cells at 21% O2 level. It had also not improved OPCs proliferation in both conditions. When cells cultured in serum-free medium at 5%O2 were plated at different densities, there were significant changes in culture characteristics. Cells plated at higher density proliferated more intensely, but the process of their maturation was significantly slowed-down. The cells seeded sparsely had more complex morphology and were able to express myelin proteins even only after 2 days in culture. Conclusions: OPC differentiation proceeds correctly through its typical stages in physiologically-relevant conditions created in vitro. Application of serum-free media and normoxic oxygen tension provides a good model to analyze active compounds OPC release during growth in well-defined conditions. Supported by NCN (National Science Centre, Poland) grant no.2014/15/B/NZ4/01875

EP30. Central amygdala circuit mediates appetitive social interactions
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Both human and animal brains are adapted to social interactions. The ability to interact with conspecifics is essential not only for survival but also for transferring emotional states of different valence. Positive social interactions are crucial for healthy development, emotional well-being as well as establishment and maintenance of adequate brain structures. Impairments in the ability to attribute positive value to social stimuli characterize several psychiatric disorders, such as autism spectrum disorder, antisocial personality disorder and schizophrenia. Social interactions are relatively well described at the behavioral level, however still little is known about neuronal mechanisms involved in these complex phenomena. To address this question, we decided to investigate elementary appetitive social interaction between rats, which were previously socially deprived to intensify social motivation. We observed, that rats which were single-housed, exhibited significantly more social behaviors during interaction compared to pair-housed animals. They also showed more 50-kHz appetitive vocalizations (USVs) than control animals. Previous studies have suggested, that social
interactions activate several brain structures connected with motivation and reward, including central nucleus of the amygdala (CeA). To identify CeA circuit activated during social interaction, we injected transgenic Venus_PSD95 rats with anterograde transport tracer to visualize active efferent projections of CeA. We observed several brain structures in which neurons receiving inputs from CeA are activated during social appetitive interactions, including ventral tegmental area, substantia nigra and dorsal raphe nucleus. Moreover, we showed that appetitive social and non-social behaviors activated partially distinct populations of cells in CeA. These results suggest that CeA contains neural circuits involved in positive social interaction.

**EP31. Optogenetically-driven activity of catecholaminergic neurons**
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Catecholamine containing neurons, such as noradrenergic (NAergic) and dopaminergic (DAergic) neurons, control many behavioral functions crucial for survival, such as arousal, attention, decision making and motivation. Development of optogenetic tools allows controlling activity of these neurons in vivo, in cell-selective and dynamic manner. We aimed to obtain specific optogenetic control of catecholaminergic neurons in vivo, thus providing an effective tool that could be used in future behavioral experiments. To achieve this goal we used genetically-modified Sprague Dawley rats expressing Cre recombinase under control of catecholaminergic neuronal marker promotor – tyrosine hydroxylase (TH). Rats were stereotaxically injected with adenoviral vector AAV-2 containing floxed Channelrhodopsin-2 (ChR2 – light-sensitive cation channel) and eYFP genes into locus coeruleus (LC) or ventral tegmental area (VTA). After proper expression of ChR2 selectively in the TH-positive neurons (3 weeks after transfection) an electrophysiological in vivo experiment was conducted (single unit recordings) to evaluate the causal relationship between optical stimulation (blue light; 473 nm, 10 mW) and alteration of catecholaminergic neuronal activity. Experimental data revealed that these neurons’ activity indeed can be altered by light, with neuronal responses changing depending on different stimulation protocols (light pulse width, number and frequency of pulses). Interestingly, light-evoked neuronal responses are limited by electrophysiological properties of the particular neuronal population, thus characteristic for DAergic or NAergic neurons. Demonstrated differentiation in neuronal responses will be helpful in choosing appropriate stimulation protocol for behavioral experiments involving optogenetics.

**EP32. Sharp-Wave Ripples-associated slow gamma oscillations show cortex region-specific alteration before and after learning in APP/PS1 mice model of Alzheimer’s disease**
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Alzheimer’s disease (AD) is the most common cause of dementia accompanied with various cognitive impairments which are related to degenerative neuronal changes in vulnerable brain regions such as the hippocampus. Increasing evidences indicate that Sharp-Wave Ripples (SWRs) – hippocampal oscillations that are important for memory formation – as well as SWRs-associated slow gamma
Oscillations which enable coordinated memory reactivation across hippocampal areas CA1 and CA3 (Carr et al, 2012) are disrupted in mice model of AD (Nicole et al, 2016). To better understand the role of SWRs-associated gamma events in memory formation we studied whether they are present not only in hippocampal networks but also in cortical regions involved in memory processing. This was tested during spatial learning in freely moving transgenic mice APP/PS1: a mice model of AD. The memory performance of 8–9 months old APP/PS1 and wild-type (WT) littermate mice was evaluated during 6 daily sessions of learning of spatial memory task. Whereas WT were able to learn the task the AD group expressed memory impairments. Then, we analyzed pre- and post-learning SWRs generated in CA1 during slow wave sleep as well as associated events recorded in cortical (PFC, ACC, RSC) and hippocampal areas (CA1, CA3). Our preliminary data show that SWRs occurrence rate, frequency, peak amplitude and duration were lower in AD compared to WT group with post-learning enhancement of occurrence rate and frequency observed in both groups. In WT group transient increase of slow gamma power during SWRs was present in addition to the CA1/CA3 also in ACC and RSC, with significant enhancement of the power after learning. By contrast, in AD group SWRs-associated gamma events were expressed in ACC and RSC with larger power increase before than after learning. These results suggest critical contribution of SWRs-associated gamma alteration in AD induced learning and memory impairments. References: Carr MF, Karlsson MP, Frank LM. Transient Slow Gamma Synchrony Underlies Hippocampal Memory Replay. Neuron 75(4): 700–713 (2012). Nicole O, Hadzibegovic S, Gajda J, Bontempi B, Bem T, Meyrand P. Soluble amyloid beta oligomers block the learning-induced increase in hippocampal sharp wave-ripple rate and impair spatial memory formation. Scientific Reports 6:22728 (2016).

EP33. Unraveling electrophysiological diversity of the rat dorsal raphe nucleus neurons with the use of cluster analysis
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Certain types of neurons exhibit characteristic electrophysiological activity. When stimulated with a current injection pulses (as in the patch clamp technique), specific types of action potential properties can be observed, e.g. threshold value, action potential shape, action potential amplitude, etc. One group of such cells is dorsal raphe nucleus (DRN) neurons. DRN region is considered one of the main sources of serotonergic (5-HT) innervation in the rat brain, however it consists of GABA-ergic and glutamatergic interneurons as well. Both classes of cells, 5-HT and interneurons, differ in electrophysiological activity parameters. Therefore, putative types of DRN neuronal cells can be distinguished during patch clamp recordings. Although the discrimination is possible mostly between 5-HT and non-5-HT cells, it is quite arbitrary. In fact, not every recorded neuron can deliver a clear representation of a type, since it was proven that particular features can overlap or that certain types of neurons co-express both serotonin and GABA neurotransmitter. It has been also reported that two electrophysiologically various classes of 5-HT cells can exist in DRN. To improve the recognition method and unravel hidden classification, statistical cluster analysis was performed. Using this multiparametric statistical tool, as this study shows, cluster analysis of 98 recorded DRN neurons reveals 4-cluster-like structure. Each cluster can be described with an individual set of parameters values. Further statistical and comparative analysis results in presumably two groups of 5-HT neurons and at least one group of interneuron cells. We believe that using this simple method can help to obtain more precise and detailed division of DRN cells subtypes.
Present knowledge about *Drosophila melanogaster* eye colour mutants describe aberrations in many aspects of their physiology and behavior. This group of mutants is a very useful tool for research on insect physiology and neurobiology. Unfortunately, focusing on holometabolous insect species gives one-sided point of view. In 2015 in the Department of Animal Physiology and Ecotoxicology (Faculty of Biology and Environment Protection, University of Silesia) two strains of eye colour mutants were isolated - *yellow* and *white* strains of *Acheta domesticus*. The house cricket (*Acheta domesticus*) is a basic hemimetabolous model insect for developmental and behavioral research. The data show that both lines contained decreased levels of ommochromic pigments. This suggests that tryptophan metabolism pathway is dysfunctional. Synthesis pathways of ommochromes are tightly connected with the metabolism of neurotransmitters (dopamine and serotonin), which affect functioning of animal nervous system. Our previous research showed that crickets mutants show differences in development and physiology. In this research, we aimed to evaluate whether mutation influence on territorial fighting behaviour of eye colour mutants (white and yellow) compare with wild type. In the round experimental chamber two males were placed. The fight was recorded and afterwards analysed in Behaview software. Fight duration was measured from the first contact until establishment, when the winner forces his opponent to retreat. The intensity of the fight was scored by using Stevenson’s scale of levels of aggression. Obtain results show differences between strains in aggressive behavior.

**EP35. Transfer of emotional information in rodents**

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Recently, we can observe a steady increase in the number of autism spectrum disorders (ASD) cases diagnosed every year. The ASD population is characterised with deficits in social interactions and communication, and presence of stereotyped behaviour. Lack of social skills is often explained with impairments of empathy, a phenomenon thought to be limited only to humans, albeit recent findings suggest that emphatic behaviours also occurs in other, non-human animals, such as monkeys, birds, rats or mice. Even though biological mechanism of this process is not yet fully understood, our knowledge in this field is expanding due to studies conducted on non-human animals. This research offers a better and more detailed insight into the pathophysiology of diseases like ASD. The simplest forms of empathy, emotional contagion, can be examined in rodents, with the use of model of between subject transfer of emotional information. In this paradigm mice are housed in pairs, one of them labelled as a Demonstrator and the second one, as an Observer. In test session, Demonstrator is subjected to aversive stimuli outside of the home cage, while Observer remains there. Then, the Demonstrator is returned to the home cage, where it can freely interact with the Observer. Ninety minutes after the start of such interaction animals were sacrificed and brain tissue was isolated. The patterns of c-Fos protein expression in the amygdala and prefrontal cortex, structures considered
relevant in controlling emotions was then assessed using immunohistochemistry methods. In case of activation of the same regions in Observer and Demonstrator, we concluded that transfer of arousal and shared representation of the stimulus occurred. In our previous experiments we have shown that C57BL/6 mice, which are believed to be normo-social, are indeed capable of transferring the emotional information, while the BTBR T + Itpr3 tf /J mice, a model of idiopathic autism, are not capable of that process. In the current study we checked whether transfer of emotional information can happen, when c57BL/6 mice is a Demonstrator and BTBR mice is an Observer. Results indicate that indeed, there is transfer of emotional information in this B6-BTBR pairs, although it is impaired compared to the B6-B6 pair.

**EP36. Spike timing-dependent plasticity in the rat’s primary motor cortex**  
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Motor cortex is involved in control, execution, learning and planning of voluntary movements and motor sequences. It’s widely accepted that learning of motor skills is based on plasticity in the motor cortex and that this plasticity occurs via NMDA receptors-dependent mechanism. Spike timing-dependent plasticity (STDP) is an event-dependent plasticity based on the Hebb’s rule. Time interval between activities of presynaptic and postsynaptic cells and order of those activities define whether strength of synaptic connection increases or decreases. Main goal of my research was to induce spike timing-dependent plasticity in the primary motor cortex. Acute slices of rat’s brain was used. Pyramidal neurons in the layer II/III was patch clamped and stimulated excitatory postsynaptic current was measured. Preliminary results suggest that STDP take place in the rat’s primary motor cortex but rules regarding this type of plasticity are changed for this brain region.

**EP37. 17β-Estradiol upregulates NTPDase2 in hippocampal gliosomes of female rats**  
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Objectives: Changes in astrocytic function may underlie the neurochemical alterations in limbic areas after oestrogen loss in adult females. Besides providing trophic and structural support to neurons, astrocytes are a major source of inflammatory mediators – extracellular adenine nucleotides. Since NTPDase2, an ecto-enzyme that preferentially hydrolyzes ATP, is expressed by rat astrocytes, we examined extracellular hydrolysis nucleotides ATP, ADP during oestrus cycle in the hippocampal glial subcellular particles - gliosomes. We also examined whether gonadal steroid hormone deprivation affects ATP and ADP hydrolysis, and role of 17β-estriadiol on NTPDase2 properties. Methods & Results: Intact adult female rats were used to assess the fluctuation in ATP, ADP hydrolysis in hippocampal gliosomes throughout the estrous cycle. Another group of female rats were submitted to bilateral ovariectomy (OVX), and three weeks after the surgery rats were treated with a single dose of 17 β-estriadiol benzoate (17βE2, 33.3μg/kg). The changes in ATP hydrolysis were not observed (proestrus, estrus and diestrus: 242.5 ±10.9, 267.3 ±10.8 and 226.4 ± 12.7 nmol Pi/min/mg, respectively) while ADP hydrolysis showed cyclic fluctuations across the estrus cycle (p<0.01). Hydrolysis of ADP was significantly higher in estrus and diestrus (54.2±4.2 and 50.4± 5.3 nmol Pi/min/mg, respectively, p<0.05) compared to proestrus (32.5±3.4 nmol Pi/min/mg). In OVX animals, we observed significant decrease in ATP hydrolysis (165.6 ± 13.1 nmol Pi/min/mg, p<0.01) compared to all three phases, while ADP hydrolysis was similar to the ADPase activity at proestrus (24.1± 1.2
nmol Pi/min/mg). Immunoblotting analysis confirmed NTPDase2 as dominant ectonucleotidase in the hippocampal gliosomes, whose relative protein abundance also decreases after OVX. Further, significant increase in the ATP hydrolysis (276.8±21.2 nmol Pi/min/mg, p<0.001) and NTPDase2 protein abundance (p<0.01) were observed in rats 24 h after the treatment, compared to OVX controls. Conclusions: These results suggest slight fluctuation of extracellular ADP but not ATP hydrolysis in the hippocampal gliosomes across oestrus cycle. ATP/ADP hydrolyzing ratio strongly argues in favor of NTPDase2, which is confirmed by immunoblot analysis. Since ovariectomy might induce astrocytic responses similar to those observed after injury and affect neuronal chemistry and morphology, our finding that 17βE2 upregulates NTPDase2 which may modulate inflammatory reactions within the hippocampus, could represent a useful therapeutic target in human disease. Acknowledgement Supported by Ministry of Education and Science, project No 173044 and 41014.

EP38. Maternal high-fat and high-sugar diet during pregnancy and lactation causes disruption in NMDA subunits protein expression in ratsâ€™ offspring brain structures
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It is well-known that pregnancy is a period when developing organism is most susceptible for environmental factors. Mothers’ imbalanced diet may be considered as one of them. Considering importance of NMDARs in various neuropsychiatric diseases, we have investigated possible influence of maternal diet on NMDAR subunits expression in rats’ offspring. Methods: Female Wistar rats were divided into 3 groups. First group – control – was fed with standard, balanced diet during whole study. Other groups were fed with special high-sugar or high-fat diet, respectively, for 5 days before coupling, pregnancy and lactation period. Offspring was separated from mothers in 21st postnatal day, divided into 28-day-old and 70-day-old group and fed with the standard diet. In appropriate time animals were decapitated and brain structures (the prefrontal cortex, the nucleus accumbens, the hippocampus and the dorsal striatum) were collected. NMDAR subunits protein expression was determined using the Western blot method. Results: Our study reveals that maternal both high-sugar and high-fat diet causes significant alternations in NMDA subunits protein expression in rats’ offspring brain structures. In rats entering adolescence most disturbances, as an overexpression of particular NMDA subunits, have been observed in the dorsal striatum, the nucleus accumbens and the hippocampus. Moreover, more changes were observed in the offspring of mothers fed a high-sugar diet. In adult animals most of disruptions were observed in the dorsal striatum, the nucleus accumbens and the hippocampus, but changes were inverse in comparison with obtained from adolescent animals. It should also pay attention to the subunit NR3A/B, whose overexpression was found in three of four analyzed structures in case of high-sugar diet. Conclusions: Our results showed that both maternal high-sugar and high-fat diet produces numerous alterations in the NMDA subunit proteins expression in offspring. This may indicate memory function disturbances, reward system function disorder and the emergence of rigid habits. The observed changes seem to be age-dependent and brain-region specific. Acknowledgments: This study was supported by the grant 2015/19/D/NZ7/00082 from the National Science Centre, Poland.
EP39 Prefrontal cortex single unit activity during extinction session of conditioned fear

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Extinction has been commonly perceived as a retroactive inhibition phenomenon, were new learning inhibits the behavioral expression of the previously learned association (Pavlov 1927; Konorski 1967). Extinction of conditioned fear bases on exposing the subject repeatedly to the stimuli that has been previously associated with the threatful event and elicits conditioned response. Behaviorally, extinction is associated with a reduction of conditioned fear response. It is well documented that dorsal (cingulate cortex, ACC and prelimbic area, PL) and ventral (infralimbic, IL) regions of the medial prefrontal cortex (mPFC) differentially regulate conditioned fear responses. Indeed, whereas PL stimulation increases fear expression, the same manipulation applied to the IL decreases fear expression. In addition IL is critical for the consolidation of the extinction memories. Despite their opposite regulation of fear behavior, until now very little is known about the possible opposite activity of those regions during the extinction of conditioned fear. To address this question, we performed single unit recordings aiming simultaneously dorsal and ventral regions, during the fear extinction. Extinction sessions were long enough to induce large reduction of fear expression upon the conditioned stimuli (CS) presentation. We hypothesised that dorsal mPFC activity would be increased at the begining of the extinction session as a correlate of fear behavior whereas ventral mPFC activity would increase at the end of the session, as the neural correlate of fear reduction.

EP40. Characterization of spreading depression using cross-correlation method

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Aim: The aim of this study was to characterize changes among signals with spreading depression and without it using cross-correlation method. Materials and Methods: The local field potentials (LFPs) were recorded from prefrontal cortex and occipital cortex in acute electrophysiological experiments. Rats were anesthetized with 2.5% isoflurane in an N2O/O2 mixture. During the two hours of LFP recording a swab soaked by 3M NaCl (group 1- without spreading depression), 3M KCl (group 2- with spreading depression) or by aCSF (group 3- without spreading depression) was applied to the occipital cortex. Cross-correlation was calculated in different frequency bands (delta, theta, alpha beta and gamma) for the signal recorded in prefrontal cortex. LFP were cutted into 10 minutes episodes. Each episode was cutted into 10 seconds fragments. For each episode we have done cross-correlation for each fragment and then calculated mean value of the cross-correlations and SEM for the present episode. Results: Cross-correlation enabled us to observed what happened in particular part of time. The most pronounced changes were observed in all frequency bands in time between 10-20 minutes and 40-50 minutes for group KCl (group with spreading depression) but not in NaCl and aCSF groups (both groups were without spreading depression). In all three groups we could observe „oscillations”. Conclusion: Cross-correlation is a good method to find and classifie differences between groups. Also this method is useful for „oscillations” detection – but depends on how the signal is devided (into smaller or bigger timezones of the signal). Paulina Urban was funded by grant of the Polish National Science Centre 2014/15/B/ST6/05082.
EP41. Cocaine-induced conditioned place preference changes the level of EAAT2 and xCT in rats brain
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Among alterations in release of various neurotransmitters caused by cocaine administration, disturbance in glutamate transmission tend to play an essential role in processes underlying addiction to cocaine. Glutamate homeostasis is provided by glutamate transporters EAAT2 and xCT. Moreover, observations in humans showed that addiction to cocaine develops only in about 20% of its users what can be also observed in laboratory animals. Taking these information in consideration we aimed to determine the expression of glutamate transporters in selected brain structures in two groups of rats: animals showing cocaine-induced conditioned place preference (addiction-vulnerable phenotype) and animals not showing cocaine-induced conditioned place preference despite prior cocaine administration in conditioned place preference paradigm (addiction-resistant phenotype). For this purpose we used male Wistar rats (n=60) which underwent 12-day unbiased conditioned place preference procedure (CPP) with cocaine (15 mg/kg; i.p.). According to the difference of time spent in a cocaine-paired chamber during test and pre-test animals were assigned to the groups: showing addiction-vulnerable (AV) or addiction-resistant phenotype (AR). After the CPP test all animals were immediately decapitated and the brain structures were isolated and frozen in -80°C until further laboratory studies. The level of the glutamate transporters was determined by Western Blot analysis. The results showed a decrease of EAAT2 in both phenotypes in prefrontal cortex. A rise in EAAT2 expression in dorsal striatum in AV and an increase of EAAT2 in hippocampus in AR were detected. No changes in xCT level were found at this stage of research. Obtained results suggest that diminished level of EAAT2 seems to be a pharmacological effect of cocaine administration and cannot serve as a factor distinguishing AV and AR. A rise in EAAT2 in dorsal striatum in AV and an increase of EAAT2 in hippocampus in AR indicate that this change may play a role in transition to compulsive drug use in those animals. On the other hand, increase in EAAT2 expression in hippocampus in AR phenotype suggest increased clearance of glutamate in this structure in animals resistant to cocaine addiction which can further alter postsynaptic excitations leading to disturbances in forming of memory circuits connecting the drug and drug-associated stimuli. Acknowledgements: The study was funded by National Center of Science under the project nr UMO-2013/11/N/NZ7/01617

EP42. Effects of GABAA and NMDA receptors disruption on the neuronal activity of dopaminergic cells
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Dopamine (DA) synthesizing neurons within ventral tegmental area (VTA) and substantia nigra pars compacta (SNc) of the mammalian brain form the core of reward and motivation system. Tonic release of DA into target structures supports animals’ basal motivation and motor functions,
while phasic increase of released DA signals reward and induces synaptic plasticity. Investigation of mechanisms responsible for developing of bursting activity has well established that presence of fully functional NMDA receptors is crucial to evoke bursting mode of activity. Beside documented engagement of phasic dopamine release in information encoding and memory consolidation, disruption in dopamine release is proposed as one of mechanisms leading to development of depressive-like behaviors. The transition between different activity modes is controlled by both excitatory (glutamate) and inhibitory (GABA) inputs, reaching dopaminergic cells. The aim of our study was to determine the effects of disruption of GABAA and NMDA receptors on electrical activity of DA cells. In our research we have used two strains of adult mice - with selective, inducible knock-out of gamma subunit of GABAA receptor (Gabrg2) and inducible deletion of NR1 subunit of NMDA receptor. Extracellular in vivo recordings of dopaminergic cells’ activity were conducted on mice under urethane anesthesia. By using iontophoretic application of GABAA receptor antagonist - bicuculline, agonist - muscimol and NMDA, we tested the responses of DA-like neurons in the ventral tegmental area and substantia nigra pars compacta of both strains. Our results show differences in baseline activity and in response to iontophoretically administered drugs between mutant and control animals. DA neurons lacking Gabrg2 had significantly increased spontaneous firing and were characterized by weaker responsiveness to GABAA receptor compounds. Oppositely, DA neurons lacking NR1 subunit of NMDA receptor had decreased basal firing and showed no response for NMDA application. Also bursting activity was significantly attenuated in comparison to control animals. Differences in effect of manipulation with NMDA and GABAA receptors is seen not only on neuronal level, but also translates into the opposite phenotypes in behavioral tests. Both presented animal strains can be used as convenient models for studying mechanisms controlling activity of dopaminergic neurons.

EP43. Neural correlates of socially transferred fear
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Emotional contagion, defined as sharing of the emotional states between individuals, is considered the simplest form of empathy. In our model of socially transferred fear we showed that a brief social interaction with a fearful cage mate (demonstrator) promotes aversive learning in an otherwise naïve rat (observer) and activates the amygdala of the observers, especially its central part (CeA). To elucidate the role of neuronal circuits in the central amygdala of the observers, we used transgenic rats expressing in behaviorally activated neurons a PSD-95:Venus fusion protein and injected with anterograde tracer. We discovered strong activation especially in the periaqueductal gray (PAG) and dorsal raphe nuclei (DRN), structures receiving dense projections from the CeA and implicated in fear and anxiety disorders. Moreover, our results showed that optogenetic activation of CeA neurons involved during interaction with recently fear-conditioned partner decreased social exploratory behaviors and ultrasonic communication. During the optogenetic stimulation rats spent more time actively exploring their environment. This pattern of behavior resembles active fear response and suggests that the CeA neurons are not only involved in classically conditioned but also in socially induced fear.
EP44. Electrophysiological characteristics of neurons in the nucleus incertus of the rat – study using multichannel microelectrode arrays
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Hippocampal theta oscillations play a critical role in numerous brain controlled functions, such as navigation in space, formation of memories or generation of different behavioral states (e.g. arousal or phases of sleep). The medial septum and diagonal band of Broca are key, forebrain structures responsible for the generation of hippocampal theta rhythm, but synchronization of this rhythm is sustained by the activating signal originating from the reticularis pontis oralis located in the brainstem. The ascending connection between these forebrain and pontine structures is polysynaptic and one of the relay stations is the nucleus incertus (NI). This GABAergic nucleus is located bilaterally in the dorsal tegmentum and adjacent to the fourth ventricle. Its main and widely described function, namely modulation of hippocampal theta rhythm, has been confirmed by experiments with stimulation or lesion of NI, which led to induction or abolishment of theta rhythm in the hippocampus, respectively. The electrophysiological characterization of NI neurons is unclear, so the main goal of this research was to fulfill this gap in our knowledge. To record neural activity, 32 channel microelectrode arrays were used. This effective method allows simultaneous recording of electrical activity of a numerous representation of nucleus incertus neurons. Extracellular recording was conducted on urethane-anaesthetized rats - under this anaesthesia two states of the brain, described as activation (theta waves in EEG) and deactivation (delta waves in EEG), were present. Results of this research indicates that neurons of the NI show activity patterns that are more complex than has been previously described. Further studies ought to be carried out in order to describe the temporal relation between the electrical activity of nucleus incertus neurons and hippocampal theta oscillations.

EP45. Electrophysiological characteristics of neurons in the nucleus incertus of the rat - juxtacellular recording-labeling study
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Nucleus incertus (NI) is a structure of large GABA-ergic cells located below the IV ventricle along the midline of the tegmentum. A subpopulation of NI neurons contains a neuropeptide, relaxin-3 (RLN 3). The relaxinergic system innervates many brain regions and is involved in stress response and behavioural activation of the animal. Recent studies revealed that nucleus incertus may be a structure involved in generation and modulation of hippocampal theta oscillations. This rhythm is associated with brain activation during stress, REM sleep or exploratory behaviours. It has be shown that there are 3 electrophysiological types of cells in the nucleus incertus. This classification was made on the basis of the firing rate of NI neurons. However, our knowledge of nucleus incertus and the mechanisms of theta rhythm generation remain poorly understood. The aim of our study is
to characterize NI neurons on the base of their electrophysiological and also biochemical properties in reference to hippocampal oscillations. All experiments were conducted on Sprague-Dawley rats under deep urethane anaesthesia which is characterized by sleep-like alternations of the brain state (activation – REM sleep and deactivation – nonREM sleep). We used the juxtacellular technique to record electrical activity and label single neurons in the NI. Firstly, the NI neuron activity was recorded during different brain states observed at the level of field potential from the hippocampus. Then a chemical marker (Neurobiotin) was iontophoretically released from the recording micropipette by applying positive current pulses (2-10 nA; 200ms, 50% duty-cycle). This procedure causes electroporation of the cell membrane and influx of released marker into the neuron. After the experiment the cell loaded with Neurobiotin was histochemically visualized and its neurochemical content (presence of relaxin-3) could be immunocytochemically determined. Our results show that in the NI there are more electrophysiological cell types than has been previously described. For example we observed NI neurons that generate action potentials in rhythmic bursts synchronized with hippocampal theta oscillations. There is also a small population of neurons with single spike activity at 4-5 Hz frequency. We propose more precise electrophysiological division of NI neurons along with biochemical identification. Identification of this neurons may help us to better understand the mechanisms underlying theta oscillations.

EP46. Chloride Ion Interaction Site in α1β2γ2 GABAA Receptor Transmembrane Domain in Deactivated State
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While numerous reports provided a substantial insight into the homomeric receptors, investigations of heteropentamers (including most commonly occurring GABAA receptors) proved considerably more difficulties. In order to address the structure and observe atom scale interaction with ions, homology model of α1β2γ2 GABAAR was build based on the glycine receptor (GlyR) structure in closed conformation with no ligands at the binding sites. As expected, the main pore constriction point was found in the middle of the transmembrane domain (TMD) at level of Leu9'. This hydrophobic ring is present also in GlyR and is highly conserved in other pentameric ligand gated ion channels (pLGICs) and is interpreted as the channel gate. In our model we found an additional constriction, but of higher radius, at the top of TMD on the level of 20' residue. Although sequences building this constrictions are not strictly conserved, but a similar pore narrowing was detected also in other channels like glutamate chloride ion channel (GluCl) in apo state or GlyR in closed state. Notably, this constriction is rich in charged and polar amino acids in other pLGICs raising a possibility that it could interact with permeant ions. To examine whether this constriction is stable in the presence of water and ion molecules in the pore a molecular dynamics simulation was performed. During the simulation the constriction radius remained unchanged, but it was not tight enough to prevent passage of water molecules. In another step, a chloride ion was docked into the top part of TMD and new simulation was run. Initial ion docking within this constriction resulted in its repetitive entrances and exits and eventually to a stable reorganization of TMD top characterized by widening of pore in this area by about 10 Å resulting in a typical closed pore profile observed in crystal structures. Interaction site within the constriction at 20' is formed by Glu270β2, Lys274α1 and Glu285γ2 with a prominent contribution of γ subunit glutamate underscoring importance of heteromeric assembly. Notably, 20' constriction profoundly differs from the 9' ring, which is symmetrically made by leucines. Obtained results are clearly indicating that, even in closed state, interactions with ion in TMD are playing an important role in shaping receptor conformation and subunit-specific contributions may be crucial for its function. Supported by NCN grant DEC-2015/18/A/NZ1/00395.
Among the viral products released by HIV-infected cells & involved in the pathogenesis of HIV-associated neurological manifestations is the soluble protein Tat. It has been reported that Tat expression/application does cause profound functional (loss of LTP) & behavioral deficits, which underlie key features of HIV associated neurocognitive disorders [1, 2]. These are likely to be occurring at the molecular level & influencing synaptic function & organization. As the loss in LTP coincided with disruptions in learning & memory, we hypothesized that Tat may modulate NMDA receptors, as the induction of LTP requires the activation of NMDA receptors & postsynaptic Ca2+ signaling [3]. To check our assumption we applied peptide Tat 47-58 (neurotoxic HIV-1 PTD) on NMDA-induced currents from isolated hippocampal neurons. All experiments were performed in accordance with the guidelines set by the National Institutes of Health for the humane treatment of animals & the Animal Care Committee of Bogomoletz Institute of Physiology. The Wistar rats (P 8-14) were anaesthetized using sevoflurane & decapitated. The NMDA receptor induced currents were measured in acutely vibrodissociated CA1 pyramidal neurons from hippocampal slices (400-500μm) using whole-cell patch-clamp technique (Vhold=-70mV) in combination with extracellular solution switches. The NMDA receptors in CA1 hippocampal neurons were activated by extracellular application of NMDA (200uM) & glycine (5 uM). Application of TAT 47-58 inhibited NMDA-receptor mediated current in a concentration-dependent manner: IC50=295±35 nM, nH=0.92±0.18. As HIV-1 Tat PTD contains rich arginine and lysine residues, different basic peptides were tested. In contrast to His9 or Lys9, the peptide Arg9 inhibits amplitude of NMDA-induced currents: IC50=9.6±0.7nM, nH= 0.78±0.03. Thus, HIV-1 PTD (Tat 47-58) inhibits NMDA currents from CA1 hippocampal pyramidal cells probably due to arginine. We can speculate that this effect underlie HIV-associated learning & memory deficits. 1. Fitting S. et al. Biol Psychiatry. 2013;73(5):443-53. 2. Behnisch T . et al. Brain Res. 2004;1012(1-2):187-9. 3. Andersen P. The Hippocampus Book. 2007.

The intraperitoneal injection or local infusion of NMDA receptor antagonists (MK-801) to the basolateral complex of amygdala (BLA) causes impairment of cognitive functions. What is more, numerous studies have shown that this effect was reversed by treatment with clozapine. The aim of our study was to verify how the electrical activity of basolateral complex of amygdala (BLA) and nucleus accumbens (NAc) changes after infusion of MK-801 and intraperitoneal injection of clozapine and whether it correlates with the behavior of rats during classical fear conditioning. This study was approved by the Local Ethics Committee. Male Wistar rats were implanted with electrodes in right NAc and right BLA and with guides in right and left BLA and then randomly assigned to two groups: AMK group (MK-801 infused and clozapine injected before acquisition sessions) or EMK group (drugs given before first three extinction sessions). As a CS we used a tone associated at the end with an electric shock. During each session of the experiment we recorded the LFPs in
NAc and BLA and behavior of rats. Number of fecal pellets was also counted. Freezing periods were established with BehaActive software (Boguszewski). Analysis of freezing duration revealed delayed extinction process in EMK group (in MK-801+Cloz experiment and previous experiment with MK-801 administration only). Administration of Clozapine before Acquisition sessions resulted in extinction and lack of differences for frequency bands between sessions in comparison to the MK-801 experiment for all analyzed time segments. Most of the differences were present during freezing periods in comparison to no freezing periods. Differences in behavior (Acquisition vs Extinction) were associated with differences in power of frequency bands between sessions, what may reflect different mechanisms of these processes.

EP49. Effect of extended treatment with typical neuroleptics on NPQ/spexin, kisspeptin and POMC mRNA expression in the rat amygdala

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Neuropeptides play an important role in the various neural pathways being able to control the wide spectrum of physiological responses. Numerous recent findings proved that antipsychotic medications may significantly affect the peptidergic signaling in the various brain structures. However, there is still no information concerning the relationship between the treatment with the classical neuroleptics and kisspeptin, proopiomelanocortin (POMC), spexin mRNA expression in the amygdaloid complex. In the current study we assessed the mRNA level of aforementioned factors in the amygdalae of animals chronically treated with typical antipsychotic drugs using quantitative Real-Time PCR method. The studies were carried out on adult, male Sprague-Dawley rats. Three groups of animals have received intraperitoneal injections with respectively control vehicle, haloperidol (2mg/kg/day) and chlorpromazine (5mg/kg/day) every day for 4 weeks (28 injections). All individuals were quickly sacrificed under anaesthesia, their whole brains were removed, then the amygdaloid complexes were precisely excised. After that the reverse transcription Real-Time PCR reactions were performed. Levels of mRNA expression were compared relative to the housekeeping GAPDH and beta-microglobulin genes. We have shown that both neuroleptics administered chronically increased the POMC mRNA expression in the amygdala that may suggest the presence of alternative pathway of their pharmacological activity. Conversely, extended drug treatment decreased the level of Kiss-1 mRNA expression supporting the hypothesis that this neuropeptide may be also involved in the control of affective processes, anxiety responses and possibly also in the pathophysiology of some mental dysfunctions. The changes in the spexin expression were also present. It may be suggested cautiously, that haloperidol and chlorpromazine may affect amygdalar neuronal populations by the modulation of the neuropeptide activity. Undoubtedly this hypothetic regulatory mechanism requires further pharmacological and neurochemical investigations. This preliminary study underlines for the first time a complex nature of potential interactions between neuroleptic action and POMC, kisspeptin and spexin signaling in the rat amygdala.
EP50. Effects of ketogenic diet on astrocytic proteins after pilocarpine administration

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Ketogenic diet is broadly studied as a method for alleviating seizures in epilepsy. Shifted balance between two modes of energy production: neuronal glycolysis and oxidative metabolism on one hand, and amino acid-based astrocytic metabolism on the other, is thought to underlie the positive effects of these diets. Several mechanisms could be in play, from altered rates of glutamate and GABA production (in favor of GABA) to downstream signaling onto molecular pathways responding to metabolic stress or energy balance. These changes result in amelioration of seizure symptoms and in decreased astrogliosis. Here, we analyzed the effects of ketogenic diet consumption on the expression of astrocytic proteins: glial fibrillary acidic protein (GFAP) and aquaporin 4 (AQP4) in rats with pilocarpine-induced status epilepticus. Thirty day old male Wistar rats were randomly assigned to normal and ketogenic diet groups and maintained on their respective diets for 30 days. At P60 a subset of these animals were given pilocarpine i.p. (250 mg/kg), behavioral symptoms were observed for 6 hours, followed by sacrifice by decapitation. The resulting experimental groups were the following: untreated controls with normal diet (CC), untreated rats on ketogenic diet (KC), pilocarpine-treated animals with normal diet (PC) and pilocarpine-treated rats on ketogenic diet (PK), with n=4 for each group. Hippocampi were dissected manually and Western blotting was used to assay GFAP and AQP4 expression. Chemiluminescent Western signal was normalized to total protein load per lane obtained from Stain-Free (UV-catalyzed trihalo reaction) signal. Between-group normalized protein levels were compared with analysis of variance (ANOVA) with Fisher’s LSD used for post-testing. Ketogenic diet itself did not significantly change the expression of either GFAP or AQP4 in comparison to controls on normal diet (CC). Pilocarpine-treated rats, on the other hand, had increased levels of both GFAP and AQP4 (PC versus CC; p<0.05 and p<0.01, respectively). Ketogenic diet seems to prevent this increase: in the PK group the levels of both GFAP and AQP4 were similar to controls (p>>0.05). Thus, we posit that ketogenic diet feeding influences the propensity of astrocytes for activation and gliosis in the acute (6h) phase after pilocarpine. This work was supported by the National Centre for Science grant UMO-2015/17/B/NZ7/02953.

EP51. Influence of naloxone injection into the pedunculopontine tegmental nucleus on feeding induced by ipsilateral stimulation of the ventral tegmental area

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The pedunculopontine tegmental nucleus (PPN) is anatomically connected with neurons in the ventral tegmental area (VTA), the initial structure of the mesolimbic dopaminergic system. The mesolimbic system mediates motivated behaviors for various survival behaviors, including feeding. The present study investigated the role of non-selective opioid receptor antagonists in the PPN (microinjection of naloxone at various doses) in the regulation of feeding behavior induced by electrical stimulation of the VTA. All rats (n = 18) were implanted with electrodes into VTA and cannulas into PPN. The behavioral model of the VTA stimulation-induced feeding response in rats was used. Latency to response was measured as a function of stimulation frequency before and after unilateral intra-PPN injection of naloxone (doses: 2.5, 5.0 and 25 μg dissolved in 0.5
µl of water; n = 6 for each doses). This experimental method allowed us to distinguish between motivational vs. motor aspects of tested reactions. Naloxone intra-PPN injections were observed to improve ipsilateral VTA stimulation-induced feeding response (Anova), which manifested as a decrease in the reaction threshold and a leftward shift of the latency/frequency curve. Lowest dose induced substantial behaviour-increasing effects accompanied by statistically significant decrease in the reaction threshold by about 16%, higher dose by about 13%, and the highest dose by about 10% compared to the control water microinjection for each experimental rat (Tukey post hoc test). Opioids receptors are one of the important factors in PPN-VTA circuits involved in the regulation of motivational aspect of food intake. Research was funded by Polish National Science Centre, Grant No.: D/NZ4/02499.

EP52. Experience dependent brain plasticity was observed in the somatosensory cortex in rat brain
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Full crossing of whiskers sensory pathway in rats brain, result in somatotopic whisker representation in the Barrel field (BF) of contralateral hemisphere. Incorporation of [14C] 2-Deoxyglucose (2DG) correlates with the metabolic rate, therefore it is successfully used for brain activity mapping. The activation of cortical areas can be visualized by following autoradiography and computer based image analysis (MCID). The metabolic activation in BF was induced by stimulation of one row of whiskers. For the implication of plasticity four rows of whiskers on the left side were trimmed for one month, whilst one row B was spared. The visualized representation of the whiskers in the barrel field varies in density, for this reason it is possible to identify an inner and an outer area of activation. The hemisphere correlated to deprivation showed wider row B representation. The experimental setup consisted of subjects with intact hemispheres and of subjects with induced phototrombotic lesion. The focal stroke 1mm +/- 300µm in diameter was placed 1mm +/- 400µm behind the barrel field. Deprivation was induced in injured hemisphere. The stimuli were set ipsilateral and contralateral to the deprivation and in the control groups were bilateral. Distinguishing and comparing the center with the surrounding area among the six different groups shows no differences in the center area (max ±3,43%; p>0.05). While the hemispheres correlated to deprivation showed a greater flexibility of the surrounding areas up to 50% more than in the control group (p<0.001). Since there are big variations in the continuity of size and metabolic rate between center and surround, it appears that the mechanisms regulating the outer and inner area plasticity might be different. The complexity of the substructures and their interactions demand additional research.
EP53. Influence of alpha frequency photic driving on visual detection thresholds
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Recent studies demonstrate that entraining the visual system using alpha frequency photic driving modulates visual detection thresholds of subsequent target stimuli (Mathewson et al, 2009, 2012; Spaak et al., 2014). However, these studies have reported apparently opposing results with regards to the time course of this effect. Mathewson et al. (2012) found peak sensitivity for a target onset inter-temporal phase with the entrainer (e.g. a target occurring when an entrainer would have appeared) and minimum sensitivity at time points that would lie mid-way between entertainers. In contrast, Spaak et al., (2014) report peak sensitivity when the target and entrainer were out-of-phase and minimum sensitivity at in-phase time points. Here we report data that indicates these apparent differences can be resolved by the different levels of spatial uncertainty with regards to the target location in the previous studies. Using a novel variant of Mathewson's paradigm,27 participants judged whether a near threshold black target presented laterally (either left or right of fixation), appeared before black annulus masks appearing around both possible target locations (80% target present). To manipulate level of spatial uncertainty, in some trials a central arrow cue pointing in the direction of (or opposite) the target location was briefly displayed prior to the onset of the target. Flicker primes were presented at both locations prior to target onset and these were either a regular 10Hz frequency in one hemifield or jittered flicker (with the same start and endpoint but a random temporal distribution in-between) in the other hemifield. The regular flicker was equally likely to be at the target or none target location. The results replicated those Mathewson and Spaak in different conditions. When spatial location of the target was congruent with the arrow prime, people were most sensitive to the target at time points in-phase with the 10Hz entrainer. This effect was not observed at these time points for the jittered entrainer. On the other hand, when the location of the target was not explicitly cued and people were best at detecting the target out-of-phase of the 10 Hz entrainer.


EP54. HRV-biofeedback: the effects of session count on psychophysiological functioning – preliminary results
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Heart rate variability (HRV) reflects the interplay between the sympathetic and parasympathetic signaling in the heart. High HRV parallels healthy psychophysiological states. Naturally occurring cardiovascular resonance between the baroreflex and respiratory sinus arrhythmia (RSA) results in increased HRV. HRV-biofeedback (HRV-BFB) based on paced breathing takes advantage of the resonance phenomenon in order to intentionally increase HRV. Due to bidirectional heart-brain interactions, such voluntarily evoked state of coherence results in improvement of health, mood,
and performance. However, literature lacks a consistent methodological approach to HRV-BFB training, as the effects of training are reported following different numbers of sessions. The current study attempts to establish the effects of HRV-BFB on psychophysiological functioning after 2 and 4 weeks of training. ECG data was collected from young healthy volunteers (experimental group N=16, control group N=9) upon 10-minute resting state before training and following the 10th and 20th session (no intervention in the control group). Both linear and non-linear indices of HRV were analyzed. Dose-dependence of changes in psychophysiological functioning are discussed. Effects of HRV-BFB intervention are presented with respect to control group.

**EP55. What does our brain listen to? Follow-up effect in EEG signal during exposition to binaural beat**
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Binaural beat is an auditory illusion which occurs when two slightly different tones, listened on headphones (one on each earpiece), give a receiver an impression of hearing a third tone. There are scientific papers a-plenty trying to prove that this illusion can help reduce stress and that it positively influences relaxation, learning process and creativity, but an overwhelming number of those works focuses only on testing the results of being exposed to a binaural beat and most of them lack a description of neural basis which causes (if causes) those outcomes. We want to present effects of our experiments in which we took a closer look at specific phenomena. Binaural beats causes interesting reaction in brain called “follow-up effect”. During the “follow-up effect” brainwaves tend to “follow” and increase frequency equal to the divergence of the tones that a receiver perceives while listening to a binaural beat. We presented various special prepared paradigms with binaural beats to group of people and measured the waves in specific range.

**EP56. Physiological Noise in fMRI**
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Physiological noise occurs when subjects breathe, move or due to their heart beats during fMRI session. These fluctuations can obscure neuronally induced signal changes (BOLD). It means that physiological noise can generate falsely positive or negative activation. Our work provides a comparison between data with correction for physiological noise and without it. We used EPI readout sequence in 3T Philips Achieva system with experimental paradigm: inferring on the intention of others. After conventional preprocessing for fMRI data in SPM12, correction for physiological noise in TAPAS PhysIO Toolbox, GLM analysis with T- and F-contrasts, we can see a difference between result without correction in PhysIO Toolbox and after that revision, but the inclusion of cardiac and respiratory regressors did not show a large improvement compared to including motion regressors only.
EP57. Gait differences between elderly and young women during task navigation

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Navigation is an ability which allows people to orientate and move in space. In order to define the position in environment people are using an external visual information and signals generated by the proprioceptive system during walking. Apart from great role of navigation in everyday life some of its changes can be treated as a marker of early brain changes in healthy aging people. Previous research showed that Alzheimer disease (AD) and Mild Cognitive Impairment (MCI) subjects gain worst results in navigation tasks and they have grater problems with walking than healthy participants despite the fact that AD does not impair the motor functioning at the early phase of disease development. Taking into account the fact that perturbation in gait can be a sensitive parameter of cognitive changes the main goal of our study was to test what kind of gait indicator is the most efficient in differentiating navigation results in group of healthy people. In our research we compared group of young (from 17 to 23) and elder (from 65 to 79) high functioning woman in their ability of navigation. The subjects were tested using Navigation Task (NT) which was divided into three phases and implemented in natural environment. During each of navigation tasks participants were equipped with motion sensors. Using this procedure gave us possibility to monitor various indicators of gait. Our analysis showed that the most sensitive indicator of differences in navigation was the effectiveness of the task (number of steps and path length) in combination with gait style (changes in gait between the following Navigation Task phases). This parameter let us predict with 83% accuracy the belongingness navigation result into the appropriate age group.

EP58. Is brain neurodynamics tied to self-control?

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Strelau’s Regulative Theory of Temperament (RTT) specifies temperament traits as basic features of personality determined by genetic factors that undergo environmental influences. Intensification of particular temperamental traits influences ones cortical arousal and activity. Complexity of the electroencephalographic (EEG) signal reflects the amount of independently processing bioelectrical generators, i.e. neuronal circuits, which underlay observed EEG activity. Estimating complexity of EEG signal in absence of task (resting-state) can be used as a psychobiological marker compatible with the RTT when studying individual differences in tonic cortical arousal. Moreover, studies conducted on twins show that characteristics of complexity of the neurodynamics are highly genetically determined. Presented study concerned relationship between resting-state EEG complexity and behavioral self-control, which is, among other temperament traits, crucial for effective, intentionally driven behavioral inhibition that results in avoiding the consequences of inappropriate behavior. The 5 minutes resting-state EEG was acquired from 28 participants (19 females) aged 19-31 (21.7 mean
± 2.9) with 64-channeled EEG system. Participants’ temperament traits were obtained using Formal Characteristics of Behavior-Temperament Inventory Modified (FCB-TI (M)). Results showed negative correlation between behavioral self-control and complexity of resting-state EEG signal in right hemisphere, right prietal, left parietal and whole parietal region. Therefore, we conclude that intensification of behavioral self-control is linked to better organization of neuronal circuits, which is beneficial for proper impulse controlling.

**EP59. Divergent thinking and Heart Rate Variability Biofeedback**

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Heart rate variability (HRV) reflects the changes in time intervals between adjacent heartbeats. It is considered to be a good indicator of control over health and psychological challenges. High HRV is associated with good health and well-being, while low HRV is related to pathological states. Moreover, HRV appears to be related to executive functioning and cognitive performance. It is possible to voluntarily increase HRV via the HRV-biofeedback technique. Aside from aiding treatment of psychosomatic disorders, this method appears to be a promising cognitive training technique. It has been previously reported to improve attention-related processes, however, its influence on creativity has not been studied in detail. The current study investigated the effects of HRV-biofeedback training on divergent thinking. Linear and non-linear measures of HRV were analyzed. Preliminary data indicate that this technique might be helpful to improve creativity.

**EP60. Electrophysiological correlates of timbre imagery**

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The principal cognitive objective of this project was to verify whether the imagined timbre is reflected on the level of event related potentials (ERPs). Previous studies on timbre imagining were of a behavioural nature or, just in one case, used fMRI imaging technique. The primary scientific objective of this project was to verify the hypothesis of how the imagining of different timbres, varying in values of the spectrum center of gravity (SCG), impact on the amplitude of the Late Positive Component (LPC), associated with imagination-related processes. Furthermore, this study evaluated the impact of the above-mentioned spectral component of timbre on the value of Auditory Evoked Potentials (AEPs) N1 and P2, during sound perception. Another independent variable taken into account by the project was expertise in music. Results of this study show that imagining sounds with high SCG is related with greater amplitude of the LPC, than in the case of imagining sounds with low value of the spectrum center of gravity. This effect was visible on channels placed above left temporal lobe. Imagining sounds, with more complex spectra, elicit lower amplitude of the N1 and higher amplitude of the P2, than imagining sounds with simple spectra. In previous studies, the P2 component was recognized as indicator of timbre processing. In this study, it was shown that the P2 amplitude in perception and the LPC amplitude in imagery are influenced in the same way by manipulation of the SCG value. This observation provides evidence in favour of the theory suggesting functional correspondence between perception and imagery. No evidence has been found that expertise in music impacts on amplitudes of the LPC and AEPs.
EP61. Auditory steady-state responses to stimulation of different duration
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The brain electrical activity can be considered a dynamical system which is able to oscillate at multiple frequencies. Activity within gamma range (30–80 Hz) frequencies is known to be important for a number of perceptual and cognitive processes. Fast repetitive auditory stimulation in gamma range elicits periodic oscillatory auditory steady-state response (ASSR) which can be recorded with electroencephalogram (EEG). The frequency of the ASSR is close to the frequency of stimulation and the greatest magnitude is observed when stimuli are presented at 40 Hz. 40 Hz ASSR power and phase precision are decreased in schizophrenia and bipolar disorder and therefore 40-Hz ASSR is increasingly used as a marker of brain function and dysfunction in neuropsychiatric disorders. However, little data exists on the optimal properties of stimulation for to reliably obtain and evaluate ASSRs. The specific impact of auditory stimulus-train duration on ASSR is far less frequently studied. The purpose of this study was to investigate how stimulus length influence properties of 40 Hz ASSR. In this experiment EEG responses of 16 participants to 500–2000 ms auditory stimuli were analyzed. The initial findings suggest ASSR properties vary depending on duration of stimulation. The results of this study point to the urge to consider stimulus duration in the future studies more carefully.

EP62. The development of a mental number line with the use of the mathematical computer game and cognitive-motor training
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Many studies confirm the benefits of cognitive training using computer games and the modern technology in education becomes more and more popular. Their results show a positive effect of such techniques i.a. on the level of mathematical skills in numeracy, recognizing numbers and transcoding different formats of numerical magnitudes and on the spatial–numerical association development. The aim of the study was to examine the effect of cognitive training with computer math game “Kalkulilo” (developed in our lab) in the development of such skills as numerosity assessing, number magnitudes comparison with the use of different formats of displayed numbers (Arabic symbols of numbers and non-symbolic dots patterns) and localization of numbers on the number line (spatial–numerical association). Twenty two children (aged 7–10) participated in the study. They were randomly divided into 4 groups: 1st group was training with “Kalkulilo” on a laptop, children in 2nd group was training with the same game but using the kinect control. The 3rd group played the control game and the 4th one was the passive control group (without any cognitive training). Training took 5 hours and was divided into 10 half-hour sessions. Before it (pre-test) and after (post-test) we measured the level of mathematical skills of participants (calculation, numbers comparison ability, numerosity estimation, knowledge of Arabic numbers, arithmetic, number line estimation) using the paper-pencil test and the computer test. The results indicate the effect of training using the “Kalkulilo” game on spatial–numerical development because it improves the accuracy of estimation the numbers location on the mental number line. This effect is particularly pronounced in the group of cognitive-motor training
(with Kinect control of movements), which further suggests this type of motor-cognitive training is more effective than standard training using only a computer. It could be concluded that the use of mathematical game training may therefore be a valuable tool not only in math education but also it could be helpful e.g. in overcoming the cognitive deficits observed in dyscalculia.

**EP63. The spatial-numerical association in the grapheme-color synaesthesia**
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Synaesthesia is a phenomenon in which some stimuli can elicit the additional experience related to the same or different modality. Its one of the most common form is the grapheme-color synaesthesia, which is manifested in color perception of numbers. Mental representations of numbers may be associated not only with colors, but also with some other attributes, such as the size, texture, or atypical spatial arrangement. The mental representations of numbers are arranged spatially on the Mental Number Line (MNL), and one of its demonstration is the SNARC (ang. Spatial Numerical Association of Response Codes) effect. It refers to the faster responses to numbers when there is congruency between the reaction side and the number position on the MNL (e.g. the right-hand responses to high magnitude numbers). The aim of our study was to investigate the SNARC effect modulation evoked by color of presented digits (1–9) in the parity judgment task in grapheme-color synaesthesia. It was assumed that in this case, colors individually assigned to mental representation of particular numbers would affect the occurrence and the profile of the spatial-numerical relations.

The experiment was divided into three blocks. The stimuli consisted of the digits 1, 2, 8 and 9. In the first block the black numbers were presented on a white background. During the second block, in the half of trials digits were displayed in colors which were corresponded to the mental synaesthetes’ representations (defined as congruent colors), while in the second half of trials the colors were randomly selected (and defined as incongruent). In the last block, stimuli were displayed in the color corresponded to the number located on the opposite end of the number line (e.g. “1” shown in the color typical for “9” in the particular case of synaesthesia). The obtained results showed that in synaesthetics both number magnitude (low-high) and its color determined the profile of the SNARC effect, however this effect was observed only in the reaction time and not in the percent of correct responses.

**EP64. The role of EEG measurement conditions in studying resting-state activity in the brain and individual differences**
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Aim: The aim of this study was to verify existence of relationship between the Big Five personality traits, temperamental traits and differences in spontaneous brain activity recorded after and before performing a cognitive task. Methods: 22 young, right-handed and healthy individuals were recruited to the study. The intensity of the personality traits was tested by Revised NEO Personality Inventory, whereas Formal Characteristics of Behavior – Temperament Questionnaire, modified
version (FCZ-KT(Z)) was used to determine the characteristics of temperament. The EEG signal was recorded using 64-channel EEG system during the four measurement conditions: (1) resting-state (RS) with eyes open, (2) RS with eyes closed before the cognitive task and (3) RS with eyes closed, (4) RS with eyes open after the task. To investigate the level of alpha waves power Discrete Fourier Transform method was used. Results: The results of this study have shown that spontaneous brain activity in different measurement conditions reveals personality and temperamental traits. Furthermore the traits that most strongly correlated with the difference in the alpha waves power in the condition with eyes open after and before the task are neuroticism and endurance. In condition with the eyes closed, positive correlations were also obtained for traits such as behavioral control and social desirability.

**EP65. Gender differences in frontal lobe hemodynamic response during cognitive task performance**

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Executive functioning is one of the main traits of higher-level cognition in humans. However, the mechanism of frontal lobes neurovascular coupling is far from being fully understood. For over four decades the Wisconsin Card Sorting Test (WCST) has been one of the most distinctive tests of prefrontal function, but clinical research and recent brain imaging data have brought into question the validity and specificity of this test because of unexplained variability of neuroimaging results across studies.¹ The aim of this work was to investigate possible gender differences in hemodynamic response obtained during computerized WCST which in turn could explain this variability by the ratio of gender in subjects cohort. Data analysis has been done with three different approaches (z-scores, max value, and lateralization index) in order to compare their sensitivity to gender effect.

**DEVICE:** CW-type 16 optode fNIR400 device, Biopac. **TEST:** We used the modified version of freely accessible computerized Wisconsin Card Sorting Test (WCST)². Participants completed 3 blocks: the main block consisted of the WCST with 128 cards; two identical control blocks with 64 cards before and after the main WCST were used. Control blocks were visually identical to the main task but with non-changing sorting rule in order to control for visual stimulation and non-executive frontal modulation related to the stimulus. Results shown above include only the main WCST block.

**ANALYSIS:** The collected data were preprocessed and converted to hemoglobin concentration with preinstalled fnirSoft. Further analysis was partially done by SPSS 20 and MATLAB 2014. Subjects: We analyzed 30 healthy 21.6 ± 2.6 year old right-handers (50% females) without diagnosed mental or cardiovascular disorders. All participants had on average 12 years of education. No significant differences between males and females in WCST performance were obtained: the average correct sorting rate was 79.90 % and 79.11 % (99 % CI), and 14.1 ± 7.4 and 14.5 ± 5.9 of perseveration errors.

fNIRS data were analyzed using two normalization methods: z-scores and dividing by maximum value within subject and within block. Task related hemispheric lateralization was calculated from filtered data without normalization. Z scores: Time series analysis show quantitative (p<0.05) and qualitative differences. No significant hemispheric differences were found. However, it does not apply for all optodes. X: Time series analysis show quantitative differences (p<0.05). However, it does not apply for all optodes. No significant qualitative and hemispheric differences were found. LI: Oxygenized, deoxygenized, and total hemoglobin – al
THEORETICAL POSTERS

TP1. Empathy and pain
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Experience of pain in biological context, is well-understood. Now, the research pay attention to psycho-social phenomenon of pain on how and when observers attend to decide, interpret, react emotionally and behave in response to another's pain. Knowing that, often with subject about pain, appears the concept of empathy. Emotional Empathy is a mechanism for quick reflecting inner states of other people, cognitive empathy can create mental representation of internal states of others and control their own emotional states, it is more complex, conscious element of empathy. The term empathy in pain has been expanded and defined as a sense of knowing the experience of another person, with cognitive, affective and behavioral components. Empathy for pain has consequences for the observer's own experience of pain, and also for observer's ability to cope with pain. Observational learning about pain, can give an information about potentially threatening situations.

TP2. The Intense World Theory - Kamilla and Henry Markram
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Autism Spectrum Disorders are developmental disorders diagnosed on the basis of behavioural deficits and defined by DSM-V and ICD 10 but the etiology of ASD remains unclear. There are several hypothesis of what constitutes the autistic brain in terms of molecular mechanisms, genetics, developmental processes, connectivity and circuitry. The most renown explanation is Baron-Cohen's theory in which the autistic brain's most notable feature is it's hypoactivity and hypococnectivity particularly in amygdala. In opposition to this view, Markrams postulate that actually autism is based on hyperactivity of connections. Their theory called Intense World Theory is an attempt of unifying data from different areas of research. Using VPA rat model of autism Markrams proved an increase of connectivity and existance of “hypercolumns” in a VPA rat brain. The theory underlines the fact of a sensory hypersensitivity and overstimulation in autistic individuals and suggests that this might be the cause of social withdrawal and impaired social functioning in autistic individuals.

TP3. I’m fine. Anosognosia after stroke
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The World Health Organization (WHO) definition of stroke is: “rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular origin”. Stroke is one of the most common factor causing physical disability and may lead to various deficits. One of them is anosognosia, which is defined as the lack of awareness of the disease’s symptoms. The term “anosognosia” was first used by Babinski in 1914 and referred to the absence of cognitive impairment awareness, visual disturbances and physical disability. Although of short duration – usually persists up to three months, some researchers claim it influences significantly patient’s recovery. Lack of awareness of disease’s
symptoms can affect different modalities such as hemiparesis or hemispatial neglect. Anosognosia patients are also hospitalized longer and after returning home show less activity in the context of daily activities. It is still unclear what location of stroke may lead to anosognosia, however it is classically connected with the damage of right brain hemisphere.

TP4. Psychological factors in treatment compliance and quality of life among psychiatric patients
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Compliance to pharmacological treatment plays a vital role in psychiatric rehabilitation and in successful management of mental illness but still, despite its effectiveness, reports indicate that up to 50% of psychiatric patients do not comply to their prescribed medications. Non compliance is a serious problem, leading to exacerbation of the underlying pathophysiology, relapse and re-hospitalization and eventually damages the patients quality of life. Taking this under consideration, understanding the phenomenon is highly important. Up until now, the focus of many studies in the psychiatric field was objective factors which might be related to medication compliance such as illness duration, medication side effects and symptoms severity. However, despite their importance, they might not be enough to understand the underlying motivational processes in treatment compliance. Given that, the first goal of the study is to understand the internal experience of mentally ill patients and how it is related to treatment compliance and quality of life. In order to do so we will present and examine a model of several subjective factors which despite their importance were never been examined together before. The factors which will be the focus of this study are: 1. insight into the illness, 2. internalized stigma, 3. feeling of shame, 4. loneliness, 5. perception of loss due to the mental illness, and 6. grief as a reaction to the development of the illness. The second goal of this study is to compare the relationships between the factors among different psychiatric diagnoses in the effort to understand whether there are differences in the way different patient population experience their illness and engage with their treatment. The study participants will be patients having a psychiatric diagnosis and taking psychiatric medications. Participants will be recruited in a time of partial symptoms remission from mental health centers in Hungary. This study is currently under the process of ethical approval and was not initiated yet. Therefore, its theoretical elements and important findings from the literature will be discussed. The important implications of this research will be highlighted and specified, mainly in terms of intervention programs which will improve patients compliance and quality of life.

TP5. Social aspects of the Sense of Agency
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Many classic Psychology experiments, such as the studies conducted by Stanley Milgram and Solomon Asch, have illustrated the extent to which our own actions can be influenced by other people. More recently, Cognitive Neuroscientist’s have demonstrated how our feeling of control over our action’s is formed via the integration of sensory cues. Nonetheless it is only recently the we have begun to understand how other people can influence the amount of agency we feel we have over both our own and group action’s at both a cognitive and a neural level. The first few studies in this area have illustrated, at a cognitive level, the affect that following or leading another’s actions can have on the experience of agency over the event. Furthermore, neuroimaging studies have indicated that different neural structure may be activated when an action is simply viewed as collaborative as compared independent. In this theoretical poster we will examine what is currently understood about how social aspects influence our sense of agency, as well as suggesting some exciting future areas of research.
TP6. Will the potassium ion really pass through that ion channel?
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Propagators are widely used in Quantum mechanics and Quantum field theory, but little has been made in regards to applications in Biology. These are mathematical objects which express the probability that some body goes from a point to the other in space in a given time. I will show here, numerically, that quantum mechanics is relevant in those magnitude orders, and then the derivation of a propagator for the potassium ion in a Coulomb field, i.e. in the ion channel. Also, I will be presenting some past results, obtained using different methods, to compare to mine. From this little application I would like to find some methods to simplify the analytical considerations for biological systems, especially in the brain, which is a hugely complicated environment to consider, particularly in the case when quantum mechanics is applied.

TP7. The effects of biometric feedback on cognitive reappraisal and emotion regulation in real-life financial decision-making
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Human judgment and decision-making are greatly influenced by emotion, both positively and negatively (Bachara, Damasio, Tranel & Damasio, 1997). Yet, the performance of high-level financial organisations, such as pension or investment firms, relies on the rationality and precision of their forecasts and subsequent decisions. As such, emotions can often get in the way, resulting in incorrect risk estimation on one end, and losing millions on the other. The present poster aims to outline the foundations of the collaboration with a major investment management corporation, currently in early stages of preparation, which will explore the effects of biometric feedback on evaluation of own decisions, emotion regulation and cognitive reappraisal in real-life financial decision-making. The poster will outline the theoretical background of biometric indicators of emotionality with relation to decision-making, as well as the brain mechanisms subserving emotion regulation and cognitive reappraisal. Past research into emotion in decision-making has found that emotions have a differential impact on financial decisions and risk estimation (e.g. fear increases risk-aversion, while anger and happiness decrease it; Lerner & Keltner, 2000). Elsewhere, a field study has shown that decision-makers who experience either too little or too much emotionality while trading tend to make bad decisions (Lo, in preparation). Cognitive neuroscience has also begun to elucidate the neural substrates of the interaction between emotion regulation and decision-making (Mitchell, 2013). Yet, it remains unknown how those findings can be directly applied to enhance high-level financial decision-making through emotion-guided biometric feedback.

TP8. So simple yet so significant: Mental Rotation Paradigm - relevance through the years and prospective implications
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In past decades, investigation of similarities or differences among both genders in terms of abilities, has given a new and interesting field of research, opening various and exciting research possibilities. Together with the development of different research methods, especially the ones rising from the side of technology, such as neuroimaging techniques, this field of research has been revived and placed in focus, once again. Several meta-analyses have showed that men achieve significantly
higher scores in tasks involving spatial abilities, despite the findings that magnitude of gender differences in spatial abilities seems to have decreased slightly in recent years, or could be eliminated through practice. Most commonly used test for demonstrating those spatio-visual skills is “mental rotation task”. This, very simple but efficient paradigm, was constructed and introduced into the field of Cognitive psychology in 1970s by Roger Shepard and Jacqueline Metzler, indicating the ability to rotate mental representations of two-dimensional and three-dimensional objects. Due to social relevance and practical implications, many paradigm variations and testing methods were applied. Over the years, this interdisciplinary topic, revealing gender-based efficiency, has occupied the attention of scientific ambience and as well among the wider public and quickly became not so rare narrative among the so called “popular psychology” but, unfortunately, due to popularity and hyper productivity, many unclear and/or insufficiently substantiated findings. Scientific relevance and contribution of this review potentially underlies in clarification and restriction of the roles of certain hormones involved in a process, inspection of the most relevant findings, close-up view to the recent findings based on imaging, revelation of biological basis and neural mechanisms or discussion of the relatively recent concepts, such as “extreme male brain”, what in final, would enable generation of definitive conclusions among numerous, but non-coherent literature and conducted studies.

TP9. Positive allosteric modulators for metabotropic glutamate receptors in treatment of schizophrenia
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Schizophrenia is a severe psychotic disorder and if untreated can lead to a significant decrease in quality of one’s life. Whilst not as common as affective disorders, (namely depression) it still torments a significant part of society (along with other psychotic disorders). The etiology of this disorder is poorly understood, though some genes and developmental disturbances have been described as risk factors of schizophrenia. Even though the primary cause is yet unknown, some theories explaining the mechanism of symptoms’ formation have been proposed. For over 50 years the most established paradigm was the dopamine hypothesis of schizophrenia. It states that the causes that stand behind the symptoms of the disorder are the abnormalities in dopaminergic transmission in the CNS, mainly the hyperactivity of mesolimbic pathway, which is commonly associated with “positive” symptoms. Although it provided a starting point for developing effective drugs, it failed to fully explain the etiology of all symptoms present in schizophrenic patients. The competitive thesis to the dopaminergic hypothesis is the glutamate hypothesis of schizophrenia, which has gained a lot of attention over the last few decades. Although the first suggestions for the importance of glutamate in schizophrenia appeared in 1960’ties, at that time there was not enough evidence to support those claims. Expansion of knowledge about CNS functioning explained the mechanisms that stand behind those reports. It appears that schizophrenia may be caused by hypofunction of the NMDA receptor. It is difficult to improve its functioning, because its stimulation is linked with excitotoxicity in the brain. This is the reason for which research today concerns not agonists of NMDA receptor, but rather other ways to reduce its hypofunction. One way of achieving this is through drugs that affect metabotropic glutamate receptors. The agonist binding site on those receptors is a highly conserved sequence so it is difficult to synthetize drugs that would have efficient selectivity. That’s why researchers today search for molecules that target other binding sites and thus may have higher selectivity while having as good clinical effects as orthosteric agonists. Those are the positive allosteric modulators – drugs that bind to allosteric site (binding site that is topographically different from orthosteric site, but it is still linked with it).
TP10. The serum zinc concentration as a potential biological marker in patients with depressive disorder
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Depression is a worldwide known illness without a proper understanding of its pathology. The lack of biological marker doesn't help in treating patients with this disease. However, several reports indicate decrease of zinc level and in some cases its deficit in clinical depression. Maybe in the future zinc will be used as a marker of clinical depression. Moreover, it is also believed that zinc may be associated with drug resistance. Currently, we are unable to state whether the shortage of this element can be one of the causes of the disease or one of its consequences, but we know that zinc increases the effectiveness of treatment with antidepressive drugs. We hope that in the future zinc will be included in the therapy of depressed patients as a marker in clinical treatment.

TP11. Diagnostic methods in Alzheimer’s Disease
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Alzheimer’s Disease (AD) is the most common neurodegenerative disorder, which causes significant decrease of memory and other cognitive processes as attention or higher cognitive processes. Currently used diagnostic protocols contain tests of amyloid-beta and tau in blood plasma, cerebro-spinal fluid, structural and functional brain scanning as well as genetic and cognitive tests. These methods allow to show both predisposition to later symptoms and control the deficits depth. However we still need methods that allow to better recognize the bases of AD and there is still no effective cure. Many countries of European Union run programs concerning AD. Studies provided under these programs showed that using various diagnostic methods is the most auspicious in order to monitor deterioration of symptoms. However, in many countries (including Poland) only part of methods is available. There are a few hypotheses about the cause of brain atrophy that results from aggregation of neurofibrillary tangles in hippocampus and other brain structures. Scientists assume that AD may be a consequence of inflammatory processes in the brain. Part of the studies show that AD may be treated as a third type of diabetes. Newer studies also showed that the amyloid-beta did not have significant influence on cognitive deficits in AD.

TP12. Why and how MDMA should be used in therapy of posttraumatic stress disorder?
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Before 1985 3,4-methylenedioxymethamphetamine (MDMA) was often used to enhance psychotherapy of depression, substance abuse, relationship problems and autism. Some new studies (Bouso et al. 2008; Mithoefer et al., 2010, 2013; Oehen et al., 2013) argue that MDMA should be brought back to psychotherapy after its criminalization. Considering that MDMA helps to achieve easier access to emotions and memories, trustfulness, higher level of empathy and self-acceptance, those researches suggest that the substance has great potential in treatment of posttraumatic stress disorder (PTSD). It creates an opportunity to replace pharmacotherapies and make existing psychotherapies more effective. Moreover, above-mentioned studies showed that probability for serious adverse events to appear during MDMA-assisted treatment is very low. Significant results
occurred in Mithoefer’s (2010) study: MDMA-assisted therapy was more effective for the subjects with treatment-resistant PTSD than non-drug psychotherapy. Follow-up study in 2013 showed that majority of the subjects who get treatment with MDMA maintained gains in symptom relief for 74 months after sessions. Although it was first randomized controlled pilot study, it gives a clue on effectiveness of MDMA-assisted therapy. Taking MDMA leads to increased activation of ventromedial prefrontal cortex (vmPFC), decreased activation of the amygdala (Gamma, 2000), increase in the release of serotonin (Green, 2003), whereas PTSD characterized by the opposite pattern of neurobiological functioning (Olszewski, Varrasse, 2005; Shin et al., 2005). In Carhart-Harris’ (2014) study conducted with healthy volunteers MDMA increased resting state functional connectivity between the amygdala and the hippocampus (decreased connectivity between those regions has been found in subjects suffering from PTSD (Sripada, 2012)). Those mechanisms presumably may explain why MDMA could be helpful in therapy. Yet there is still lack of researches showing how MDMA affects brains of patients with PTSD, therefore further studies and discussion in this field are needed. Here we provide neurobiological reasons to use this drug in therapy and try to introduce a way of preventing possible dangers of MDMA-assisted treatment.

TP13. Physiological mechanisms of the respiratory influences on the brain functioning
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The role of peripheral physiological processes in the generation of affective experience (peripheral feedback) has been studied widely since William James and Oscar Lange proposed their peripheral theories of emotions. In recent decades knowledge about the role of the peripheral feedback was extended by the advances in our understanding of the afferent neural pathways and the central regions (such as Insular cortex), that integrates afferent signals and allowed perception of the physiological state of the body (interoception). Breathing is one of the peripheral processes that are modulated by emotions and can influence the affective state. A well known mechanism of peripheral respiratory feedback on affect is hyperventilation (breathing that excess metabolic demands). It results in hypocapnia, an increased sympathetic output and brain hypoxia, it also upregulates affective arousal and is considered as part of defense responses. Recently Lehrer and Gevirtz proposed a physiological mechanism that may be responsible for downregulation of affective arousal when the respiratory rate decreases. It is the resonance between respiratory sinus arrhythmia and baroreflex. As a result, the amplitude of blood pressure oscillations rises, and the activity of baroreflex is increased. An increase in baroreceptors stimulation exerts an inhibitory influence on the higher centers of the CNS, through modulation of the activity of nuclei in hindbrain, midbrain and diencephalon, which regulate arousal of the CNS. Supported by National Science Centre, Poland, grant nr UMO-2015/19/N/HS6/01601

TP14. Deep Brain Stimulation as a promising method for Bipolar Disorder
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Bipolar Disorder (BD) - one of the main causes of disability among young adults it’s a psychiatric disorder characterized by at least one manic or hypomanic state and recurrent episodes of depression. Occuring of treatment resistant depression during the course of BD is very common. One of the most known ways to treat unremitted depressive patients is electroconvulsive therapy (ECT) causing many side effects impairing patients’ everyday life. There is a new promising method which has
been tested also in BD – Deep Brain Stimulation (DBS). It is associated with a single neurosurgical low – risk procedure resulting in immediate antidepressant effect in most patients with BD I and II. Neuroimaging studies confirmed the area of sIMFB as the most DBS reactive. sIMFB combines reactivity of all previously tested regions. Further studies on the stimulation of sIMFB in selected groups are needed to introduce a new, safe treatment for refractory depression in the course of BD.

TP15. Nusinersen and Spinal Muscular Atrophy (SMA) - clinical trial experience
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Spinal muscular atrophy (SMA) is an autosomal recessive motor neuron disorder that affects approximately 1:10 000 births. It is classified into clinical types 0-4 differentiated by age of onset and highest motor function. SMA is caused by the deletions or the loss-of-function mutations in a Survival Motor Neuron 2 gene (SMN2) located on chromosome 5q13. Nusinersen, previously known as a ISIS-SMNRx, is an antisense oligonucleotide designed to alter splicing of SMN2 mRNA, in patients with childhood SMA. Drug delivery to the central nervous system remains a key to enable development of therapies to treat diseases based on the known genetic mechanisms. Lumbar puncture, generally safe and straightforward procedure, is performed for diagnostic and therapeutic purposes in children and infants. A clinical trial which 28 patients 2 to 14 years of age with type 2/3 spinal muscular atrophy participated in intrathecal injections will be discussed. Lumbar punctures were successfully performed in children with spinal muscular atrophy in the initial nusinersen clinical studies. Nusinersen was well-tolerated when given as a single intrathecal bolus injection to children with SMA and no safety concerns were identified. The favorable profile from the first human clinical study of nusinersen in children with SMA should be an encouragement for further development of SMA treatment. Presented is a succinct review of the PubMed available literature data about SMA treatment.

TP16. The activity of neurotransmitters in the course of Autism Spectrum Disorder
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Autism Spectrum Disorder (ASD) is diagnosed in 1 out of 68 newborn child. First symptoms are noticeable in 6-month old children and many of them are persistent throughout life. Main ASD dysfunctions are communication impairments, problems with expressing of emotions and non-verbal behaviours as well as presence of stereotypical behaviours. The poster will focus on brief report on how scientists connect neurotransmitters system impairments and ASD. Serotonin (5-HT) is endogenous amin located in the blood platelets, intestine cells and in the brain (raphe nuclei and pineal gland). In the brain 5-HT inhibits the regulation of central nervous system, nevertheless it also plays an important role in emotional processes. A highly replicated result in people with ASD is hiperserotonemia, which is elevated 5-HT concentration in their blood. What is more, 5-HT concentration may be even 70% higher in comparison to 5-HT level in blood in neurotypical people and is observed in more than 30% of patients with ASD [1]. In turn, acetylcholine (ACh) in the body activates muscles, and is a major neurotransmitter in the autonomic nervous system. In the
brain, ACh has variety of effects upon plasticity or arousal. Post-mortem brain analysis indicate that ACh receptors present in people with ASD have almost half less activity to agonists in comparison to receptors present in neurotypical people [2]. The other neurotransmitter, dopamine (DA) is active in the brain in several pathways. One of them plays a major role in reward-motivated behaviours, the others are involved in motor control and in controlling the release of hormones. The brain imaging (using PET) indicates that some brain structures (e.g. prefrontal cortex) in people with ASD have lower dopaminergic receptor activities than these present in neurotypical people [3]. This work was funded by Polish National Science Centre, Preludium grant: 2014/15/N/NZ4/04844.


TP17. Does the serotonergic system impairments affect the development of psychiatric disorders?

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Serotonin (5-HT) regulates many fundamental aspects of physiology and behaviours including gastrointestinal functions and mood [1]. The influence of 5-HT on the organism is strictly connected with the interaction between this neurotransmitter and the specific serotonergic receptors. Peripheral 5-HT is synthesized and stored in the enterochromaffin cells found in the gastrointestinal tract crypts as well as in the platelets. In the central nervous system, fibers which are immunoreactive to 5-HT are widely distributed. However, the cells which produce 5-HT are found only in the restricted areas of the brain, namely raphe nuclei groups which exist from the midbrain to the medulla. The poster will focus on brief report on how psychiatrists as well as neuroscientists connect the serotonergic system and the most common psychiatric disorders. Both children and adults with Autism Spectrum Disorders have hyper serotoninemia which is elevated 5-HT concentration in their blood. What is more, 5-HT concentration may be even 70% higher in comparison to 5-HT level in neurotypical people and is observed in more than 30% of patient with ASD [2]. In turn, adult patients with depression very often have elevated concentration of the 5-hydroxyindoleacetic acid (5-HIAA; the main metabolite of serotonin) in their cerebrospinal fluid. On the other hand, brain imaging indicates lower activity of serotonergic receptors (e.g. 5-HT1A) in patients with depression [3]. Furthermore, the serotonergic system impairments were also found in the ADHD. Genetic research indicates that inappropriate DNA methylation, for instance in the serotonin transporter gene (SLC6A4, 5-HTT) may elevate the probability of the disorder diagnoses [4]. This work was funded by Polish National Science Centre, Preludium grant: 2014/15/N/NZ4/04844.

TP18. Will your mobile phone understand your emotions? Affective computing approach to emotion recognition
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We seem to be entering an era of enhanced digital connectivity where human-computer interaction (HCI) is becoming increasingly essential to our daily lives. For the development of such communication systems, the most challenging aspect is emotion recognition. The poster provides the intellectual framework for affective computing concerns multidisciplinary knowledge background such as affective neuroscience, psychology, cognitive and computer sciences to quantify emotions. A practical look is taken at which available monitoring methods can be used to acquire physiological indicators of emotion, recognize, model, understand and respond to it. Since this approach to affective human-computer interaction is not advanced yet, the emphasis is placed on challenging research topics in order to develop such systems.

TP19. Every part matters: how interactions between NMDA receptors, Ca²⁺ channels and acetylcholine shape synaptic plasticity in CA1 pyramidal neurons
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Synaptic plasticity, the ability of synapses to change the strength of the signals they transduce between neurons, historically has generally been attributed to NMDA receptor (NMDAR) mediated Ca²⁺ influx and subsequent activation of kinases or phosphatases. Kinase activity has been found to result in signal enhancement, called long-term potentiation (LTP), while phosphatase activity results in the opposite, long-term depression (LTD). Conversely, converging lines of evidence showed that voltage-gated Ca²⁺ channels (VGCCs) produce large Ca²⁺ transients as well [1]. In addition, neuromodulation by acetylcholine (ACh), which has been shown to affect the induction of synaptic plasticity [2], however, the detailed description of the modes of action is lacking. In this computational study, using NEURON 7.4, a detailed multi-compartmental model of a hippocampal CA1 pyramidal neuron [3] and an NMDAR-dependent synaptic plasticity model [4], we aim to investigate two questions: whether NMDARs are sufficient as a Ca²⁺ source to account for experimental data on synaptic plasticity and how does neuromodulation by acetylcholine (ACh) affect NMDAR-dependent plasticity induction? We report that, under basal ACh levels, theta burst pairing (TBP) causes LTP in proximal spines (<170μm), but LTD in distal ones. When the cell was exposed to saturating levels of ACh, proximal spines displayed a stronger LTP, but distal spine LTD switched to LTP even stronger than that induced in basal spines. Based on these results, we predict that VGCCs in CA1 pyramidal neuron are necessary for synaptic potentiation in distal spines under basal ACh, as no studies known to us have reported LTD in distal spines induced by TBP. Also, we attribute switching of LTD to LTP in distal spines to ACh mediated inhibition of A type potassium current, which promotes back-propagation of action potentials and, by increasing spine Ca²⁺ influx via NMDARs and R-type VGCCs, allows formation of LTP. To summarize, by combining validated mathematical models, we report on putative mechanisms of how NMDARs, VGCCs and ACh juxtapose in shaping the rules of synaptic plasticity. 1. Sabatini BL, Oertner TG, Svoboda K (2002) Neuron 33, 439–452. 2. Buchanan KA, Petrovic MM, Chamberlain SE, Marrion NV, Mellor JR (2010) Neuron 68, 948–963. 3. Saudargiene A, Cobb S, Graham BP (2015) Hippocampus 25, 208–18. 4. Rackham OL, Tsaneva-Atanasova K, Ganesh A, Mellor JR (2010) Front Synaptic Neurosci 21, 1–12.
Pedophilia is a disorder of sexual preferences undoubtedly evoking huge social concern. Unfortunately, there is still not enough studies that exhibit the neural mechanisms underlying this disorder. Existing researches using the functional Magnetic Resonance Imaging exhibited the opposite response of the limbic system in patient group with pedophilia compared to controls (Sartorius, et. al., 2007). Pedophiles showed increased amygdala activation during the viewing pictures of children in swimsuits and reduced activation while the photographs of adults were presented. The phenomenon of pedophilia can be also explained by the decrease amygdalar volume and closely related structures (Schiltz, et. al., 2007). What is more, the reduced activation of the hypothalamus, periaqueductal gray and dorsolateral prefrontal cortex in pedophilic patients during visual erotic stimulation indicates that there might be neural correlates contributing to abnormal sexual preferences, which means the lack of sexual interests toward adults (Walter, et. al., 2007). Presented studies indicate that pedophilia is associated with alterations in brain structures responsible for emotions and manifests as a difficulty of impulse control at the behavioural level. For this reason this is an important problem that should be taken into consideration in future researches in order to develop better diagnostic and therapeutic methods. References Sartorius, A., Ruf, M., Kief, C., Demirakca, T., Bailer, J., Ende, G., ... & Dressing, H. (2008). Abnormal amygdala activation profile in pedophilia. European Archives of Psychiatry and Clinical Neuroscience, 258(5), 271-277. Schultz, K. (2007). Brain pathology in pedophilic offenders. Arch. Gen. Psychiatry, 64, 737-746. Walter, M., Witzel, J., Wibking, C., Gubka, U., Rotte, M., Schiltz, K., ... & Northoff, G. (2007). Pedophilia is linked to reduced activation in hypothalamus and lateral prefrontal cortex during visual erotic stimulation. Biological psychiatry, 62(6), 698-701.

Intelligence, often defined as the ability to perceive, analyze and adapt to the environmental changes, however examined by teams of researchers for decades, still remains challenging to measure. Estimating the level of intelligence in sighted people seems to be demanding and definitely not an easy task. An even greater challenge is to measure this quality in the blind. The Wechsler Adult Intelligence Scale (WAIS), which is one of the most popular IQ tests, consists of two scales: verbal and non-verbal. However it is a commonly used IQ test, it is not suitable for the blind due to its reliance on visual stimuli in non-verbal scale. In response to the lack of well-established tools, estimating the intelligence level, the Cognitive Test for the Blind was developed (Nelson, Dial and Joyce, 2002). The test is based on the verbal scale of WAIS, however the correlations between those two are not satisfying. With the development of neuroimaging techniques and achievements in experimental psychology scientists started to look for neurocorrelates of intelligence, for example by measuring the accuracy of performance in a popular n-back task while subjects underwent fMRI scans. Working memory, which this task attempts to measure, refers to a limited capacity system responsible for maintenance and online manipulation of information that gets to the cognitive system. The study conducted by Conway, Kane and Engle (2003) showed evidence that so called general intelligence strongly correlates with the above mentioned memory system (referred to as working memory) in the sighted people. Since the working memory can also be tested independently of the given sensory
input it can prove to be an excellent tool to measure the working memory capacity which can serve as a neurocorrelate of intelligence in the blind. Up to now many research suggests that cognitive functioning (such as i.e. short memory etc.) in the blind is significantly better than in the sighted participants in contrast to the results obtained in the classic paper-pencil intelligence tests when comparing sighted and blind individuals (Cohen, Voss, Lepore, Scherzer, 2010; Röder and Rösler, 2003). The proposed study attempts to answer the question how to measure intelligence in the blind using the above mentioned combinations of tools and paper-pencil tests.

TP22. Interhemispheric interactions in cortical plasticity
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Communication between hemispheres is possible thanks to the structures of white matter called commissures. In the brain of placental mammals three commissures can be distinguished – hippocampal commissure, anterior commissure and corpus callosum. The latter is the biggest and best developed in this group of mammals. Corpus callosum consists approximately of 200 million fibers connecting homologous and non-homologous areas in both hemispheres. Fibers connecting specific cortical areas travels through different parts of corpus callosum. Well propagated researches on lateralization of the brain should base on examining functions of interhemispheric interactions. In that context we can state that lateralization allows for processing information in each hemisphere without the interference from the opposite one – if so, the interhemispheric connections, especially corpus callosum in placental mammals could play a role in promoting intrahemispheric processing and further lateralization by inhibiting one hemisphere during performing tasks. On the other hand, some tasks demand the ability to share and consolidate information between two hemispheres. In that case the facilitative character of interhemispheric interactions can be expressed. Despite experiments conducted so far little is known about the exact role of interhemispheric connections in the communication between hemispheres during routine activity or in the cortical plasticity. The aim of this work is to present the current state of knowledge about the evolutionary origin and anatomical structure of interhemispheric connections, currently collected data and theories concerning their functioning (excitatory or inhibitory nature). The conclusion of this work will be an attempt to determine the current state of knowledge about the influence of interhemispheric interactions on cortical plasticity. This issue seems to be crucial in the context of rehabilitation after unilateral brain damage. Disharmony in the cooperation between hemispheres caused by the damage may affect the plasticity of the brain cortex and it can lead to the impair of restoration of lost functions.

TP23. Effect of NMDA and GABA synaptic properties on the resting state oscillations in a computational model of EEG
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Computational modeling studies offer a powerful tool to understand intrinsic mechanisms of gamma frequency alterations in schizophrenia. In this study, we employ a model of a spiking neural network to investigate the influence of the input noise characteristics and NMDA, GABA synaptic properties on the resting state oscillations that can be potentially used for schizophrenia research. The network is composed of 800 pyramidal neurons (PCs), 150 regular-spiking interneurons (RSIs) and
50 fast-spiking interneurons (FSIs) (Spencer in Frontiers in Human Neuroscience 3, 2009). All cells are randomly interconnected and have recurrent connections between each other. The background activity in the cortex is represented by a Poisson noise input to network cells (PCs, RSIs and FSIs) at 4 Hz, 25 Hz and 100 Hz. As the frequency of the Poisson noise input increases, the frequency of the network oscillations increases as well. To generate synchronous oscillations at 40 Hz in neuronal populations of PCs, RSIs and FSIs, network synaptic weights have to be readjusted for 4 Hz, 25 Hz and 100 Hz Poisson input conditions. The alterations in NMDA and GABA synaptic conductances have a profound effect on the network activity. Reduced decay time constants of NMDA and GABA synaptic conductances lead to the increased frequency of the network resting state oscillations. Reduced synaptic connectivity and NMDA receptor hypofunction that are proposed mechanisms underlying the decreased gamma frequency oscillations, can be investigated using computational modelling approach.

TP24. MTHFR polymorphism and depression: exploring the mechanisms
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Methylenetetrahydrofolate reductase (MTHFR) catalyses the conversion of 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate, a cosubstrate for homocysteine remethylation to methionine. Two of the most investigated MTHFR coding gene single nucleotide polymorphisms (SNPs) are C677T and A1298C, which in a number of previous studies have been associated with occlusive vascular disease, neural tube defects and other severe conditions. Number of growing evidence is describing the connection of not only homozygous, but also heterozygous mutations with such psychiatric conditions as schizophrenia and affective disorders. Depression continues to be a prevalent diagnosis among European citizens and by some estimates up to 40% of Caucasian population may be carriers of some kind of MTHFR gene polymorphism, therefore exploring the molecular mechanisms afflicted by these mutations may seem to be crucial for developing novel biological markers and effective treatments. As both folate cycle and methionine cycle seem to be disrupted by MTHFR mutations, their implications may lead to such conditions as homocysteine accumulation resulting in hyperhomocysteinemia and excitotoxicity or S-adenosylmethionine (SAMe) deficiency that can be linked with reduced catecholamine catabolism and lower activity of DNA methyltransferases (DNMTs). Moreover, modern Western diet consists of products fortified with folic acid (Vitamin B9) to prevent fetal neural tube defects during pregnancy, which on the contrary may be a negative factor affecting the well-being of MTHFR mutants.

TP25. Ultrasonic vocalization as a useful tool in research of autism spectrum disorders
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Autism spectrum disorders (ASD) are a class of neurodevelopmental disorders characterized by persistent deficits in social behavior and communication across multiple contexts, together with repetitive patterns of behavior, interests, or activities, which begin in childhood and persist into adulthood. In this common and heterogeneous neuropsychiatric conditions, language deficits may range from a complete lack of intelligible speech and severe delays in language acquisition to reduced conversational skills for many reasons. Ultrasonic vocalization (USV) is thought to be implicated in social communication. Isolation-induced USV are increasingly used as a measure for the quantification of communication deficits in mouse models of neuropsychiatric disorders.
characterized by social and communication deficits such as autism. Rodent USVs are a vital tool for linking gene mutations to behavior in mouse models of communication disorders, also neonatal USVs were interpreted as a communicative behavior. USVs are composed of various call types although their meanings are not yet clear. Mice with autism, including BTBR mice, exhibit abnormal patterns of USV emissions. Animal models of autism include assessing of different behaviors. Among them USV is rarely considered. Literature search revealed only 27 papers. This review summarizes the current knowledge.

TP26. Neuroenhancement
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“Neuroenhancement” refers to the targeted enhancement and extension of cognitive and affective abilities in healthy subjects based on the understanding of their underlying neurobiology. Currently the trend is being increasingly represented by the media as a desirable possibility. In reality, most contemporary strategies for neuroenhancement – comprising invasive and non-invasive brain stimulation and pharmacological manipulation – remain in their infancy. The aim of the poster is to provide the summarization of current state of scientifically-proven methods for cognitive abilities’ enhancement, which could be used for performance improvement by students and white-collar workers.

TP27. Neuromyths and psychomyths in Polish schools
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Internet and popular articles may discredit the world of scientific research. Incorrect interpretation of scientific data, cognitive distortions present in the free transmission of data, or emotional attitude leading to the duplication of distortions may be reasons grounding of false beliefs. Consequently, there is a formation of myths which leads to improper attitudes. Inveterate and false beliefs of neuroscience and cognitive psychology can cause improprieties in education and teaching. It is often incorrect transfer of information from the public lore. Popular myths in science may also affect the decision-making bias in the selection of therapy, treatment, teaching methods and conduct in everyday life. Other authors show that the myths commonly exist in the population, contributing to the spread of false beliefs and severity of stereotypes. Myths in education pose a threat in the teaching process. False beliefs of neuroscience and psychology are a problem, especially if they occur in school practice. The research, conducted by other scientists, has shown that in other populations of teachers (for example in the UK, China, the Netherlands, Greece and Turkey), myths in the field of neuroscience are common. However, there is lack of data, provided from other authors, connected to the universality of myths in Poland. In our occurrence, we would like to present the first survey of such kind of research in Poland, with the participation of teachers in the statement for the research of other authors engaged in similar work. Our goal is to identify the most common misconceptions and myths about the brain and its functioning, brain relations with the psyche, and the practice of psychology. In our study involved 270 teachers from Polish schools. We controlled socio-demographic factors, age, gender, work experience, education level and specialization of teachers. Preliminary results of this group reveal a similar level of common beliefs of teachers in Poland as in other cultures.
TP28. Foreign language and the brain
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The area of foreign language learning and its impact on brain is yet to be wholly discovered and fully understood. The other way around is not very different as there is not a whole lot of theories on how the brain influences the foreign language learning process in adult brain. With many statistically significant results one may feel the need for a summary of recent studies on the matter, especially considering the fact that not always do those studies come to the same conclusion. A series of studies show that learning a foreign language has a direct impact on brain's areas related to language learning. Some of them state that higher proficiency or performance comes in pair with bigger brain activity in the gyrus and makes it structurally more liable to change. It seems probable that white matter’s plasticity in the brain is an important factor when it comes to foreign language learning.

TP29. Is your hippocampus under too much stress? The connection between glucocorticoids and memory
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When a situation is interpreted as stressful, cortisol, steroid hormone, is released as a result of the hypothalamic-pituitary-adrenal (HPA) axis activation. Studies (McEwen et al., 1968; Reul & de Kloet, 1985) showed the link between hippocampus and stress. In both animal and human hippocampus, there are two types of glucocorticoid receptors. Studies of the effects of stress confirm the connection between glucocorticoid levels and cognitive performance (Lupien, 2016). Different clinical population such as Cushing syndrome patients (Starkman, Gebarski, Berent & Schteingart, 1992), elderly people with HPA dysfunction (Lupien et al., 1998, 1995; 1994) or major depression (Vythilingam et al., 2002) have associated level of glucocorticoids with hippocampal volume. These studies suggest the correlation between hippocampal volume, level of glucocorticoids and patients results in spatial and verbal memory tests.

TP30. Neural substrates of metaphor processing
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Metaphors are crucial component of human cognition. The ability to create and understand abstract relations between phenomena is a foundation of higher level thinking. We use metaphors in our everyday communication, in art, in public discourses. It’s one of our mind’s most potent tools of making sense out of reality. I would like to present introduction to the subject of neural substrates of metaphor processing – how we understand metaphors, what are different factors that impact metaphor processing and creating new expressions. What is an impact of metaphors on the process of learning and divergent thinking. I will shortly highlight the main points of the most interesting research from the field of neuroscience (mostly using fMRI) and cognitive science to provide a better insight into the mechanisms of metaphorical thinking and what does it tell about our cognition.
TP31. Dietary restriction ameliorates neurodegenerative phenotype in mice model of Alzheimer’s disease
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Human life expectancy has been steadily increasing for at least a century which has lead to growing rate of age-related diseases. Dietary restriction without malnutrition has consistently been shown to not only extend lifespan in various animal models, but also delay the onset of age-related diseases. Most common experimental approaches to dietary restriction are caloric restriction (CR) and intermittent fasting (IF), of which Every Other Day Feweding regimen (EODF) is most frequently used. There have been studies on effects of CR on different organisms, notably rodents, rhesus monkeys and humans, including its impact on neurodegenerative disorders and age-related functional decline of the nervous system. Although underlying molecular mechanisms of dietary restriction are not yet completely clear, they appear to be highly evolutionarily conserved. Possible mechanisms involve modulation of key metabolic regulator proteins and pathways, improved stress response and antioxidant protection, upregulation of neurotrophic factors, attenuated inflammation and enhanced mitochondria biogenesis. Dietary restriction has been shown to improve learning and memory, as in novel object recognition test and cotextual fear conditioning in mice model of Alzheimer’s disease described below. Different molecular mechanisms of such effect has been proposed, one of which, supported by another study described below, is enhanced BDNF expression which regulates basal level of neurogenesis in dentate gyrus and promotes survival of newly generated neurons.

TP32. Use of dual-task paradigm in neurorehabilitation
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Everyday life often requires simultaneous realization of two or more tasks. Efficient and flexible performance under such conditions depends on processes of behavioural control, attention resources and proficiency. Those processes, responsible for controlling human behaviour, are called „executive functions”. Researches have proved that dual-task paradigm (DTP) is useful in improving multitasking skills and often leads to reduction of interference effect by action automatization (Ruthruff, Johnson, Van Selst, 2006). DTP requires simultaneous realization of two different tasks. Combination of cognitive and motor tasks exhibits most satisfactory results in training executive functions. Moreover, the association between cognitive functioning and physical activity have been found (Beauchet et al., 2009). Existing rehabilitation programmes for neurological patients (such as Neuroforma or DynamicCognition) use this paradigm to efficiently train and improve their cognitive and motor skills - for example planning or problem solving with balance maintenance.

TP33. Structural neurobiology of Tau protein in tauopathies
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Tau protein is a microtubule associated protein that is found mainly in axons of neurons in the central nervous system. However, disruption of proteostasis which is cellular homeostasis of proteins, may trigger a cascade of pathological events leading to Tau dysfunction. Factors that influence the change of Tau include altered post-translational modifications (in particular hyperphosphorylation), failure of protein quality control mechanisms (including proteasome and autophagy impairment)
and altered interactions with molecular partners. As a result, Tau loses the ability to bind and stabilize microtubules in over 20 neurodegenerative diseases, collectively known as tauopathies. These include Alzheimer’s disease, hereditary FTDP-17, progressive supranuclear palsy, corticobasal degeneration, Down’s syndrome. In the course of these diseases Tau forms amyloid aggregates which are highly ordered protein assemblies rich in β-sheets. Such Tau structures can be deposited as paired helical filaments (PHFs), which constitute neuropathological lesions known as neurofibrillary tangles (NFTs). In tauopathies, polymorphic aggregates of Tau are found in neurons and glia, affecting diverse brain regions. The structural diversity of pathological assemblies of Tau has been proposed to be responsible for characteristic neuropathological consequences, including toxic effects and ability to cause specific neuronal dysfunction and degree of dementia. It has been postulated that a structural change of physiological Tau into aberrant structures, that accumulate in human brains during pathogenesis of tauopathies, has prion-like features. Indeed, conformational transformation of physiological protein molecules into pathological structures characterizes prions – infectious agents responsible for prion diseases (e.g., kuru, Creutzfeldt–Jakob disease, bovine spongiform encephalopathy). However, Tau aggregates do not cause epidemics in humans like true prions. As the matter of the fact, they should not be called ‘prions’ but prionoids. The last term emphasizes toxicity of amyloid assemblies and excludes their infectivity. Distinguishing prion and non-prion aspects in Tau amyloidogenesis may provide better understanding of molecular basis of tauopathies.