SESSION II

CENTRAL NERVOUS SYSTEM NEUROPHYSIOLOGY PLASTICITY OF NEURAL FUNCTION

Thursday (September 16, 2021; 11:00 – 13:55)
Thursday (September 16, 2021; 14:20 – 18:05)

Chair:

Prof. JOANNA LEWIN-KOWALIK,
Department of Physiology, Medical University of Silesia, Katowice, Poland

Dr. hab. ADRIAN SMEDOWSKI
Department of Physiology, Medical University of Silesia, Katowice, Poland

DETAILED SESSION II SCHEDULE

Opening lectures (Thursday, September 16; 14:20 – 16:50; *virtual stream B*)

S2.L1 ELECTRICAL COUPLING OF OPTIC NERVE AXONS - A NOVEL MODEL OF GAP JUNCTIONS' INVOLVEMENT IN OPTIC NERVE FUNCTION. **A. Smedowski** (Department of Physiology, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland).

S2.L2 BETWEEN RETINA AND BRAIN: PATTERN ELECTRORETINOGRAPHY. **D. Pojda-Wilczek[1], K. Gibinski[2]** ([1]Department of Ophthalmology, Faculty of Medical Sciences in Katowice, Poland, [2]University Clinical Centre, Medical University of Silesia in Katowice, Poland).

S2.L3 THE ROLE OF HUMAN ANTIGEN R (HuR)/ABNORMAL VISUAL SYSTEM-LIKE 1 (ELAVL1) IN AGE-RELATED OCULAR PATHOLOGIES – UPDATING THE PUZZLE. **M. Amadio** (Department of Drug Sciences, Section of Pharmacology, University of Pavia, Pavia, Italy).

S2.L4 NEUROVASCULAR CROSS-TALK IN RETINAL DISEASES – MODELS OF DIABETIC RETINOPATHY. **M. Pietrucha-Dutczak** (Department of Physiology, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland).

S2.L5 CAN WE REGULATE PERINEURONAL NETS AFTER SPINAL CORD INJURY? AN INSIGHT FROM GENE, PROTEIN EXPRESSION AND WFA LABELING. **M. Skup, K. Grycz, A. Glowacka, B. Ji, O. Gajewska-Wozniak** (Group of Restorative Neurobiology, Nencki Institute of Experimental Biology PAS, Warszaw, Poland).

Oral presentations (Thursday, September 16; 16:55 – 18:05; *virtual stream B*)

S2.L6 MECHANISMS OF OXIDATIVE STRESS IN THE RAT HEART IN A ROTENONE MODEL OF PARKINSON'S DISEASE. **O. Gonchar, O. Klymenko, T. Drevytska, V. Nosar, L. Bratus, I. Mankovska** (Bogomoletz Institute of Physiology, National Academy of Science of Ukraine, Kiev, Ukraine).

S2.L7 INFLUENCE OF BONE MARROW-DERIVED MESENCHYMAL STEM CELL THERAPY ON CCL2, CCL19 AND CCL20 LEVELS IN MINIMALLY CONSCIOUS STATE PATIENTS. **W. Czelejewska, E. Sinderewicz, W. Maksymowicz, K. Jezierska-Wozniak** (Department of Neurosurgery, Laboratory of Regenerative Medicine, School of Medicine, Collegium Medicum, University of Warmia and Mazury in Olsztyn, Olsztyn, Poland).

S2.L8 DROSOPHILA BRAIN REWARD SYSTEM AND POSSIBLE CONSEQUENCES FOR UNDERSTANDING THE HUMAN PLEASURE. **J. Dvoracek[1,3], D. Kodrik[1,2]** ([1]University of South Bohemia, Ceske Budejovice, Czech Republic, [2]Institute of Entomology, Biology Centre, CAS, Ceske Budejovice, Czech Republic; [3]Psychiatric Hospital Cerveny Dvur, Czech Republic).

S2.L9 PHYCHOPHYSIOLOGICAL, HORMONAL, AND RECEPTOR CORRELATIONS OF GENDER AND INDIVIDUAL DIFFERENCES IN PAIN SENSATION. **I. Kvachadze[1], M. Apkhazava[1], M. Tsagareli[1,2]** ([1]Tbilisi State Medical University, Tbilisi, Georgia; [2]Beritashvili Center for Experimental Biomedicine, Tbilisi, Georgia).

Session summary

Poster session (Thursday, September 16; 11:00 – 13:55; *virtual stream D, interactive*)

S2.P1 A NEW LOOK AT THE EXISTENCE OF THE INTERACTION OF THE AMYGDALA WITH THE VISUAL SYSTEM. **K.H. Miryusifova[1], A. Allahverdiyeva[1], N. Huseynova[1], E. Panachova[1]** (Institute of Physiology, Baku, Azerbaijan).

S2.P2 INHIBITION OR STIMULATION OF SHELL NUCLEUS ACCUMBENS CHANGES INTRAVESICAL PRESSURE AND CARDIOVASCULAR PARAMETERS IN WISTAR RATS. **R. De Carvalho[1], B. Antonio[1], N. Dsouki[1], B. Do Vale[1], P. Aronsson[2], L. De Luca Jr[3]., M. Sato[1]** ([1]Department of Morphology and Physiology, Centro Universitario FMABC, Santo Andre, SP, Brazil, [2]Department of Pharmacology, Institute of Neuroscience and Physiology, Sahlgrenska Academy, University of Gothenburg, Gothenburg, Sweden, [3]Department of Pathology and Physiology, Dentistry School, Sao Paulo State University (UNESP), Araraquara, SP, Brazil).

S2.P3 BIDIRECTIONAL EFFECT OF THE EXTREMELY LOW-FREQUENCY ELECTROMAGNETIC FIELD (50 HZ) ON BDNF LEVEL. **A. Klimek, H. Kletkiewicz, A. Siejka, M. Klimiuk, J. Maliszewska, M. Jankowska, A. Nowakowska, J. Wyszkowska, M. Stankiewicz, J. Rogalska** (Department of Animal Physiology and Neurobiology, Faculty of Biological and Veterinary Sciences, Nicolaus Copernicus University in Torun, Poland).

S2.P4 EARLY-LIFE STRESS AFFECTS PERIPHERAL AND BRAIN RESPONSE TO IMMUNE CHALLENGE IN FEMALE RATS. **A. Solarz, I. Majcher-Maslanka, J. Kryst, A. Chocyk** (Maj Institute of Pharmacology, Polish Academy of Sciences, Department of Pharmacology, Laboratory of Pharmacology and Brain Biostructure, Krakow, Poland).

S2.P5 COMPARATIVE ANALYSIS OF THE INFLUENCE OF EPIPHYSIS AND SUPRACHIASMATIC NUCLEUS OF HYPOTHALAMUS ON VISION FUNCTION. **U. Hashimova, E. Panahova, X. Miryusifova, A. Alahverdiyeva, N. Huseynova** (Institute of Physiology named after A.I. Garayev of ANAS, Baku, Azerbaijan).

S2.P6 CONFIRMATION OF THE INFLUENCE OF AMIGDALA ON THE FUNCTIONS OF THE VISUAL ANALYZER STRUCTURES IN AMIGDALAR EPILEPSY. **A. Alahverdiyeva, U. Hashimova, E. Panahova, X. Miryusifova, N. Huseynova** (Institute of Physiology named after A.I. Garayev of ANAS, Baku, Azerbaijan).

S2.P7 THE ACTIVATED MICROGLIA IN HIPOCCAMPUS AS A CHARACTERISTIC OF STREPTOZOTOCIN INDUCED MODEL OF ALZHEIMER DISEASE IN RATS. **J. Dunacka, G. Swiatek, I. Majkutewicz, P. Matulewicz, B. Grembecka, W. Glac, D. Wrona** (Department of Animal and Human Physiology, University of Gdansk, Faculty of Biology, Gdansk, Poland).

S2.P8 EFFECT OF SEROTONIN, ADRENALINE AND DOPAMINE ON THE FUNCTION OF THE VISUAL SYSTEM STRUCTURES. **N. Huseynova, U. Hashimova, E. Panahova, X. Miryusifova, A. Alahverdiyeva** (Institute of Physiology of ANAS A.I. Garayeva, Baku, Azerbaijan).

S2.P9 INFLUENCE OF CAFFEINE ON THE GENE EXPRESSION OF PROINFLAMMATORY CYTOKINES AND THEIR RECEPTORS IN THE HYPOTHALAMIC-PITUITARY UNIT. **M. Wojcik[1], M. Tomczyk[1], J. Bochenek[1], D. Tomaszewska-Zaremba[1], A. Antushevich[1], A. Krawczynska, A. Herman[2], A.P. Herman[1]** ([1]The Kielanowski Institute of Animal Physiology and Nutrition Polish Academy of Sciences, Poland, [2]Faculty of Health Sciences, Warsaw School of Engineering and Health, Warsaw, Poland).

S2.P10 THE CB_1 RECEPTOR ANTAGONIST REDUCES THE PRESSOR RESPONSE OF ANGIOTENSIN II AND ANGIOTENSIN 1-7 INJECTED INTO PARAVENTRICULAR NUCLEUS OF THE HYPOTHALAMUS (PVN) IN CONSCIOUS NORMOTENSIVE AND HYPERTENSIVE RATS. **K. Minczuk, B. Malinowska** (Medical University of Bialystok, Bialystok, Poland).

S2.P11 THE EFFECT OF NIACIN, VITAMIN B3, ON THE β-AMYLOID-ASSOCIATED PROCESS OF NEURODEGENERATION. **A. Litwiniuk[1], M. Kalisz[1], L. Martynska[1], M. Chmielowska[1], A. Domanska[1,2], W. Bik[1]** ([1]Department of Neuroendocrinology, Centre of Postgraduate Medical Education, Warsaw, Poland, [2]Department of Physiological Sciences, Warsaw University of Life Sciences (SGGW), Warsaw, Poland).

S2.P12 SWIM TRAINING AMELIORATES OXIDATIVE STRESS IN THE SPINAL CORD OF ALS MICE. **K.P. Dzik[1], D.J. Flis[1,2], Z.K. Bytowska[2], M.J. Karnia[1], W. Ziolkowski[2], J.J. Kaczor[1]** ([1]Gdansk University of Physical Education and Sport, Gdansk, Poland, [2]Medical University of Gdansk, Gdansk, Poland).

S2.P13 THE EFFECT OF BENZO[A]PYRENE ON OXIDATIVE STRESS IN CHICKEN EMBRYOS BRAIN. **R. Muchacka, L. Kolodziejczyk, G. Formicki, A. Gren** (Institute of Biology, Pedagogical University of Krakow, Krakow, Poland).

S2.P14 NEUROPHYSIOLOGICAL STUDY OF DISORDER AND RECOVERY OF SPATIAL MEMORY IN AN EXPERIMENTAL MODEL OF ALZHEIMER'S DISEASE. **E. Panakhova, U. Hashimova, K. Javadova, I. Galandarli, Kh. Miryusifova** (Institute of Physiology, Baku, Azerbaijan).

S2.P15 EFFECT OF DIMETHYL FUMARATE ON DISORDERS OF THE OLFACTORY BULB NEUROGENESIS IN THE STREPTOZOTOCIN-INDUCED RAT MODEL OF ALZHEIMER'S DISEASE. **E. Kurowska, I. Majkutewicz, J. Rucinski, D. Myslinska, K. Sawicka, N. Piekarczyk** (University of Gdansk, Department of Animal and Human Physiology, Gdansk, Poland).

S2.P16 EFFECT OF PREBIOTICS SUPPLEMENTATION ON SOCIAL BEHAVIOUR AND PLASMA TUMOR NECROSIS FACTOR-α LEVEL DISTURBANCES IN HIGH- AND LOW-RESPONDERS RATS WITH CENTRAL AMYGDALA HYPERACTIVATION. **J. Rucinski, E. Kurowska, N. Piekarczyk, D. Myslinska, I. Majkutewicz** (University of Gdansk, Department of Animal and Human Physiology, Gdansk, Poland).

S2.P17 IMPROVED MOTOR FUNCTION AS A RESULT OF THE INFLUENCE OF MINOCYCLINE ON MOTOR CORTEX NEURONS IN CORTICAL MODEL OF PHOTOTHROMBOTIC ISCHEMIC STROKE IN RATS. **K. Pawletko[1], A. Grajoszek[1,2], H. Jedrzejowska-Szypulka[1]** ([1]Department of Physiology, School of Medicine in Katowice, Medical University of Silesia, Katowice, Poland, [2]Department of Experimental Medicine Medical University of Silesia, Katowice, Poland).

S2.P18 CHANGES IN THE RESPONSIVENESS OF THE RAT DORSOMEDIAL HYPOTHALAMUS TO DIFFERENT METABOLIC CONDITIONS UNDER HIGH-FAT DIET. **A.M. Sanetra, K. Palus-Chramiec, L. Chrobok, J.S. Jeczmien-Lazur, J.D. Klich, M.H. Lewandowski** (Jagiellonian University in Krakow, Krakow, Poland).

S2.P19 MODULATION OF PAIN IN BRAIN LIMBIC AREAS: ROLE OF OPIOID AND CANNABINOID SYSTEMS. **N. Tsiklauri[1], N. Tsagareli[1,2], I. Kvachadze[2], M. Tsagareli[1,2]** ([1]Beritashvili Center for Experimental Biomedicine, Tbilisi, Georgia; [2]Tbilisi State Medical University, Tbilisi, Georgia).

S2.P20 MINOCYCLINE AFFECTS SPLEEN T AND B LYMPHOCYTES PERCENTAGE IN STREPTOZOTOCIN-INDUCED MODEL OF ALZHEIMER'S DISEASE IN RATS. **G. Swiatek, J. Dunacka, W. Glac, B. Grembecka, I. Majkutewicz, D. Wrona** (Departament of Animal and Human Physiology, Faculty of Biology, University of Gdansk, Gdansk, Poland).

S2.P21 EFFECT OF KETOGENIC DIET ON NEURODEVELOPMENTAL REFLEXES. **W. Kosiek, Z. Rauk, Z. Setkowicz-Janeczko** (Jagiellonian University, Krakow, Poland).

S2.P22 ARCHITECTURE OF A FUNCTIONAL SYSTEM OF THE SAGITTAL BALANCE MAINTAINING. **A. Goncharova** (Kharkiv National Medical University, Kharkiv, Ukraine).

S2.P23 EFFECT OF PROCAINE BLOCKADE OF THE VENTRAL TEGMENTAL AREA ON THETA RHYTHM INDUCED BY PHARMACOLOGICAL ACTIVATION OF THE PEDUNCULOPONTINE NUCLEUS. **A. Piwka, J. Orzel-Gryglewska, A. Walczek** (University of Gdansk, Department of Animal and Human Physiology, Gdansk, Poland).

S2.P24 MET-ENKEPHALIN INVOLVEMENT IN THE PROTECTION OF CEREBELLAR AND FRONTAL CORTEX IN VAGOTOMIZED RAT. **K. Pierzchala-Koziec[1], M. Wieczorek[2], A. Kobrzycka[2], P. Napora[2]** ([1]Department of Animal Physiology and Endocrinology, University of Agriculture in Krakow, Poland, [2]Department of Neurobiology, University of Lodz, Lodz, Poland).

S2.P25 FREQUENCY-DEPENDENT PLASTICITY OF SPONTANEOUS ACTION POTENTIALS WITHIN IDENTIFIED LYMNAEA'S NEURONS. **Z. Seval, A.V. Sidorov** (Belarusian State University, Minsk, Belarus).

S2.L1

ELECTRICAL COUPLING OF OPTIC NERVE AXONS – A NOVEL MODEL OF GAP JUNCTIONS' INVOLVEMENT IN OPTIC NERVE FUNCTION

A. SMEDOWSKI

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Retinal neurons are considered to be part of the central nervous system, resulting in lack of their spontaneous regeneration in response to damage. Glaucoma, a progressive optic neuropathy, is thought to be the main cause of severe visual impairment or permanent vision loss. Connexins are important channel proteins that form gap junctions connecting neighbor cells, including neurons. Despite of their undoubted importance in cell homeostasis, in neurons they may promote spreading of apoptotic insult, leading to secondary neurodegeneration. The presence of previously unknown GJs (electrical synapses) between optic nerve (ON) axons, which directly connect axons within bundles in the ON head potentially accelerates signal transduction along the ON and allows modulation of the signal passage from the retina to the brain. By creating crosswise conduction within bundles of the ON, it could possibly allow bypass of local damage within axons. We hypothesize that density and conductivity of these synapses may be crucial with respect to the susceptibility of the ON to having different impairments develop into symptomatic pathologies. We showed that transient chemical blocking of ON electrical synapses slows down visual signal conduction. In the case of axonal structural or functional impairment, the signal could possibly be passed crosswise *via* GJs to the neighboring axon; thus, the preservation of the syncytial structure of the ON can prevent the blockage of visual information propagation. This finding could have substantial implications for understanding of the pathogenesis of various optic neuropathies and identifies a new potential target for a therapeutic approach.

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S2.L2

BETWEEN RETINA AND BRAIN: PATTERN ELECTRORETINOGRAPHY

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Visual pathways start off with a retina. Retinal ganglion cells (RGC) are the last level of retinal cells. T heirs neurits form long optic nerves and transmit action potential from the eyes to the next station, lateral geniculate nucleus. Pattern electroretinography (PERG) is unique examination of RGC function. Two main waves P50 (positive) and N95 (negative) of PERG reflect function of central and pericentral RGC, respectively. In this way early localization of visual disturbances origin is possible. Abnormalities of P50 wave are attributed to macular diseases while incorrect N95 wave points diagnostic procedures at optic neuropathy. Unfortunately, the procedure is not simple. It requires not only expensive equipment and single use electrodes but also patient's good cooperation and experienced staff. Much easier is to get photopic negative response (PhNR ERG), the new modification of full field flash electroretinography. Abnormal PhNR indicates optic neuropathy but the clinical use of this response is still under consideration.

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S2.L3

THE ROLE OF HUMAN ANTIGEN R (HUR)/EMBRYONIC LETHAL, ABNORMAL VISUAL SYSTEM-LIKE 1 (ELAVL1) IN AGE-RELATED OCULAR PATHOLOGIES - UPDATING THE PUZZLE

M. AMADIO

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Increasing evidence suggests that loss of RNA homeostasis is a central feature in many pathological states, including eye diseases. Gene expression is controlled at post-transcriptional level by several factors (e.g. RNA-binding proteins, coding and non-coding RNAs) playing in concert to determine the fate of a given transcript. Among mammalian RNA-binding proteins, the ELAVL (embryonic lethal, abnormal visual system-like) family is a masterpiece of gene expression regulation by affecting RNA metabolism from splicing to translation. The ubiquitous member of this family, HuR/ELAVL1, controls the expression of genes with a key function in physio and pathological contexts. Alterations in HuR/ELAVL1 levels and/or function have been found in some cellular and animal models of age-related ocular diseases. Although the picture is far to be completed, intriguing findings suggest HuR/ELAVL1 involvement in the aetiopathology and its potentiality as a therapeutic target in eye diseases.

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S2.L4

NEUROVASCULAR CROSS-TALK IN RETINAL DISEASES – MODELS OF DIABETIC RETINOPATHY

M. PIETRUCHA-DUTCZAK

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Diabetic retinopathy (DR) is one of the most common complications of diabetes leading to vision impairment. Among patients with diabetes, the prevalence of DR is 35.4% and is higher in those with type 1, compared with type 2 diabetes. A high glucose level alters several cellular functions, such as intracellular calcium level, NADPH oxidase activity and the signalling of nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB). Hyperglycaemia stimulates the production of free radicals and reactive oxygen species (ROS) which are the main cause of oxidative stress leading to retinal vasculature damage. Vascular pathology in DR is characterised by alterations in the integrity of retinal capillaries and their occlusion, vascular leakage, subsequent neovascularization, and retinal haemorrhages. Diabetes in a rodent model is associated with an elevated apoptosis ratio in the retina, decreased numbers of ganglion cells (RGC) and a reduction in retinal nerve fiber layer thickness. Furthermore, the expression of intermediate filament glial fibrillary acid protein (GFAP) increases in Muller cells, which is a common marker for neural degeneration. It is very difficult to point out only one factor causing RGC death in DR. Many related mechanisms correspond to RGC loss, such as glutamate accumulation and toxicity, reduced expression of neurotrophic factors, signalling pathway impairment and increased production of proinflammatory cytokines. There is currently a growing body of evidence indicating that the damage of RGC appears before vascular changes and clinical signs of DR. Moreover, RGC apoptosis is preceded by synaptic neurodegeneration and dendritic retraction of these cells. Because dendritic abnormalities occur prior to RGC loss, identifying dendrite pathology can be treated as an early sign of neurodegeneration. Various animal models of diabetes have been established to improve understanding of the pathophysiology in diabetes and its complications such as nephropathy, retinopathy and neuropathy. Zucker diabetic fatty rats, BioBreeding Diabetes-Prone rat, streptozotocin rats/mouse or nonobese diabetic mouse are commonly used diabetic animal models. These animal models are of great importance in basic as well as in preclinical research. It is not only helping in understanding the disease mechanism of diabetes but also in evaluating new therapies with curative potential.

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S2.L5

CAN WE REGULATE PERINEURONAL NETS AFTER SPINAL CORD INJURY? AN INSIGHT FROM GENE, PROTEIN EXPRESSION AND WISTERIA FLORIBUNDA AGGLUTININ (WFA) LABELING

M. SKUP, K. GRYCZ, A. GLOWACKA, B. JI, O. GAJEWSKA-WOZNIAK

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In the adult nervous system, extracellular matrix (ECM) may be both dispersed in the neuropil and well-organized in a form of lattice-like neuronal envelopes, called perineuronal nets (PNNs). PNNs are composed of hyaluronan which creates a scaffold and of aggregating chondroitin sulfate proteoglycans (CSPGs) which are essential for formation and stabilization of PNN structures. In the spinal cord the most elaborated PNNs encapsulate motoneurons (MNs) by unsheathing soma and proximal parts of dendrites, forming a "perisynaptic barrier" controlling neural communication. The peri-motoneuronal net may be of essential importance for stabilization of neuronal connections in conditions of prolonged, self-sustained activity, characterizing α-MNs. Injury to the spinal cord reveals the other face of CSPGs: their overexpression in the scar contributes to pathology, by limiting fiber regrowth from the site of injury and impeding recovery of motor functions. The attempts to stimulate neuronal circuits to grow fibers and reorganize them assume processes loosening or decomposing the expanded web. We shall review our studies which demonstrated that moderate, long-term locomotor training that activates the entire spinal network is capable to enrich MN innervation, and improve locomotor function after spinal cord transection (SCT) (Macias *et al*., 2009, Skup *et al*., 2012; Gajewska-Wozniak *et al*., in prep.). In search for the molecular underpinnings of those effects we asked whether locomotor training can influence CSPGs metabolism at the protein level, and the structure and distribution of the PNNs around MNs after SCT. We shall demonstrate that SCT at the thoracic level leads to (1) a significant increase of mRNA levels and to a lesser extent protein levels of neurocan, phosphacan, brevican, aggrecan, and NG2 but not Hapln1 protein linking the net, in thoracic and to a lesser extent in lumbar segments of the spinal cord at subacute (2-nd week) and chronic (5 weeks) postinjury; (2) a decrease in their transcripts in MNs located in the lower lumbar segments, at 2 weeks post-injury. Surprisingly, in MNs, a denervation-elicited suppression of transcription was not reflected by the levels of CSPGs proteins, which were maintained at control level around these cells. We shall show also that locomotor training, which was not effective in modulating CSPGs central core protein levels around MNs, was a stimulus to down-regulate markedly a density of PNNs which was significantly increased after SCT. These results point to the possibility that training applied to spinal animals leads to modification of PNNs density through modifying chondroitin sulfate glycosaminoglycan side chains of central domains. To verify this possibility studies on enzymes catalyzing glycosamino-glycan assembly are needed.

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S2.L6

MECHANISMS OF OXIDATIVE STRESS IN THE RAT HEART IN A ROTENONE MODEL OF PARKINSON'S DISEASE

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Oxidative stress (OS) is caused by an imbalance in the redox state of the cell either by overproduction of reactive oxygen species (ROS), predominantly in dysfunctional mitochondria, or by impairment of the antioxidant systems. Accumulating evidence suggest that OS may play a significant role in pathogenesis of neurodegenerative diseases including Parkinson's disease (PD). In rodents, rotenone administration reproduces several features of PD, including nigrostriatal dopaminergic degeneration and typical alpha-synuclein-positive intracytoplasmic inclusions in the brain. Whereas evidence for increased ROS production and impaired antioxidant defenses in PD brain is reasonably strong, relatively few studies to date have established the mechanisms of OS in other organs and tissues, in particular, in PD heart. At the same time, PD is a well-recognized risk factor for developing heart failure, and cardiovascular complications are the important cause of PD-related morbidity and mortality. The identification of a number of PD-related genes that are strongly associated with mitochondrial function (*PINK 1, DJ-1, Parkin*) further adds weight that mitochondrial dysfunction with resultant OS is a primary event in PD pathogenesis. This study was therefore designed to investigate the biochemical and genetic mechanisms of OS developing in the rat heart in a rotenone model of PD. It was found that prolonged systemic subcutaneously rotenone administration significantly increased the H_2O_2 production, protein oxidative modification and the intensity of lipid peroxidation in rat heart mitochondria. Rotenone administration caused a significant increase in the MnSOD activity with concomitant decrease in the activity of GPx ($P < 0.05$). Simultaneously, we have found an increase in GSSG level, a decrease in GSH content, and at that the ratio of reduced to oxidized form was 2 times less than the control value ($P < 0.05$). These changes were accompanied by an increase in MnSOD and a decrease in DJ-1 protein synthesis. It was also established *DJ-1* gene deficiency, whereas the level of *PARK2* mRNA was increased ($P < 0.05$). In addition, we studied the hypoxia inducible factor (HIF) gene expression, which regulates transcriptional activation of several genes responsive for oxygen transport, glycolytic metabolism, angiogenesis, and apoptosis. Therefore, HIF activation can serve as an indicator of mitochondrial homeostasis. It was found that the *HIF* (subunits 1;2;3-α) mRNA levels in the rat heart were reduced compared to the control value ($P < 0.05$). So, increased ROS production and impaired antioxidant defenses in the heart under rotenone administration could result from the established *DJ-1* gene and DJ-1 protein deficiency. Moreover, we can assume that a decrease in HIFs gene activation may have an effect on mitochondrial functional state as well.

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S2.L7

INFLUENCE OF BONE MARROW-DERIVED MESENCHYMAL STEM CELL THERAPY ON CCL2, CCL19 AND CCL20 LEVELS IN MINIMALLY CONSCIOUS STATE PATIENTS

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Minimally conscious state (MCS) is a severe disturbance of consciousness, in which minimal behavioral evidence of self or environmental awareness is demonstrated. MCS may arise from traumatic brain injury or structural brain lesions, which are often accompanied by an excessive release of inflammatory factors by activated microglia and astrocytes, leading to neuroinflammation. Lack of the effective therapy of MCS has highlighted the need to look for alternative treatment methods, such as mesenchymal stem cells (MSC) therapy. These cells display high secretory activity and have been shown to possess immunomodulatory properties which can modify neuroinflammation. Therefore, the aim of the study was to assess the impact of bone marrow-derived mesenchymal stem cell (BM-MSC) administration on chosen chemokines - CCL2, CCL19 and CCL20 - levels in CSF and plasma of MCS patients. Nine patients aged 19–45 years, remaining in MCS for 3–14 months, were given BM-MSC three times at two-month intervals. The samples of CSF and plasma were collected before the treatment (control) and after the first and second BM-MSC administration. Relative expression levels of selected chemokines were determined by dot-blot method using Human XL Cytokine Array Kit. Obtained data revealed alterations in chemokine contents both in plasma and CSF after BM-MSC administration. The increased level of CCL2 and decreased levels of CCL19 and CCL20 were observed in CSF after cell administration, compared to the control values. In plasma, only CCL19 level was lower after the therapy. Our data suggest that BM-MSC treatment may be involved in the modulation of chemokine signaling in MCS patients.

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S2.L8

DROSOPHILA BRAIN REWARD SYSTEM AND POSSIBLE CONSEQUENCES FOR UNDERSTANDING THE HUMAN PLEASURE

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Disorders of the brain pleasure system are one of the promising topics of interdisciplinary study. The core problem of the disorder (dysregulation of the brain reward system) is being investigated by neuroscience in various animal models. The fly *Drosophila melanogaster* is a common laboratory model for studying the principles of neural network functioning. When studying the brain reward system, Drosophila is very attractive model because its relatively well-arranged brain and precisely described genome. Moreover, it can be beneficial that using Drosophila brain we do not encounter so many complex concepts with unlimited meanings (e.g. emotions, feelings, consciousness). The main problem in interpreting the study of the human brain is the complexity and ambiguity of concepts and functions. Until recently, 'pleasure' was perceived as a function of the brain of mammals and was seen as a manifestation of higher brain functions (as part of emotional circuits) or as a manifestation of complicated neural networks. In our contribution, we first clarify the current knowledge of the Drosophila reward system, emphasizing that 1) the brain regions involved in associative learning and reward functions are surprisingly complex, although the fly is a relatively simple and short-lived organism, 2) its brain almost certainly has a system that creates motivational drive (similar to the 'wanting'component of reward function in higher animals), 3) there are indications of the possible presence of the hedonic component of reward or its evolutionary precursor. Further, we mention several possible inspiring moments for understanding the human brain system, and possibly for general modeling of the reward function. Reward brain function appears to be 1) based hierarchically; 2) not organized, but operating rather with functions distributed among other brain networks; 3) its individual parts can be independent of each other and work in parallel; 4) reinforcement processing of a specific stimulus with the desired behavior can be rather multilevel. This way of understanding the pleasure system would also result in another comprehension of its disorders: e.g. 'addiction' could not be perceived only as a distortion of the mesolimbic dopaminergic system or hedonic systems of endogenous opioids/endocannabinoids, but also as a generalized disorder from cortical to cellular level, with the need for corresponding generalized interventions.

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S2.L9

PHYCHOPHYSIOLOGICAL, HORMONAL, AND RECEPTOR CORRELATIONS OF GENDER AND INDIVIDUAL DIFFERENCES IN PAIN SENSATION

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Most psychophysiological studies of experimentally induced pain revealed increase pain sensation in females compared with males, as well as variations in pain sensation over different phases of the ovarian-menstrual cycle (OMC) in females. The largest group of receptor proteins that deal with thermal and mechanical pain sensation is the subfamily of the transient receptor potential (TRP) channels and its most studied representative TRPV1 - polymodal sensory transducer activated by a diverse variety of stimuli, including heat (>40°C), mechanical pressure, acids, vanilloids (e.g., capsaicin), gingerol and endo-cannabinoids. Among the structures of the endogenous opioid system, a major player is the mu-opioid receptor (MOR), activated in the process of interaction with endogenous or exogenous opiates. Our results revealed that males show significantly higher heat pain threshold and mechanical pain tolerance than females in both phases of the OMC. Mechanical pain threshold, mechanical pressure threshold, cold pain threshold, and heat sensation threshold are insignificantly higher in males than in females in both the follicular phase and luteal phase of the OMC. The luteal phase of the OMC compared with follicular, females revealed significantly lower degrees of heat and mechanical pain thresholds, also mechanical pain tolerance, as well as nonsignificant lower degrees of cold pain and heat sensation thresholds. In males, degrees of heat, cold, mechanical pain thresholds, mechanical pain tolerance significantly positively correlate with free testosterone and MOR levels, significantly negatively - with TRPV1 levels. In females significantly positive correlation revealed between degrees of mechanical pressure, pain thresholds and tolerance and the follicular stimulating hormone (FSH) level in follicular phase of the OMC, as well as progesterone level in the luteal phase of the OMC; significantly positive correlation revealed between cold pain threshold degree and MOR level in both phases of the OMC; also a significant positive correlation between heat pain interphase decrease degree and TRPV1 interphase increase degree, as well as progesterone level in the luteal phase of the OMC. Thus, there is a significant correlation between the threshold of thermal and mechanical pain in men with psychological indicators, and the absence of such correlation in women is important for the development of gender and personalized methods of pain relief and the role of individual psychophysiological characteristics for perception and assessment of pain.

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S2.P1

A NEW LOOK AT THE EXISTENCE OF THE INTERACTION OF THE AMYGDALA WITH THE VISUAL SYSTEM

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We showed it for the first time in electrophysiological experiments that amygdala has a regulatory effect on the perceptive function of the visual system. Amygdala takes part in the regulation of visually controlled behavior and carries out the identification and discrimination of the visual image. The amygdala is known to be involved in identifying not only anxiety and fear, but also pleasure. It has been established that amygdala multidirectional effect on the visual system structures along the parvo- and magnocellular pathways are reconsidered. Reliable data obtained are a prerequisite for revising existing views of ‘independence’ and ‘parallelism’ of these pathways and is a new approach to understanding visual perception. These pathways are opposite and reciprocal to each other. These findings regarding the important regulatory multidirectional role of Amygdala in brain cognitive function have been confirmed in many papers by other scientists. The interneurons in the lateral amygdala and basal amygdala are physiologically distinct populations and suggest they may have differing roles during associative learning: basolateral amygdala contributes to a variety of behavioral patterns. Jhaveri *et al*. established the evidence for newly generated interneurons in the basolateral amygdala of adult mice 2020. It was found that the amygdala has a control effect on the visual system function.

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S2.P2

INHIBITION OR STIMULATION OF SHELL NUCLEUS ACCUMBENS CHANGES INTRAVESICAL PRESSURE AND CARDIOVASCULAR PARAMETERS IN WISTAR RATS

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Neuroanatomical studies have shown that rostral regions of the dorsomedial shell nucleus accumbens (NAcc) project to the lateral preoptic area (LPA). Injections of angiotensin-(1-7) into the LPA evokes a huge increase in intravesical pressure (IP). It is still unknown whether the shell NAcc has any role in micturition control or not. This study focused to investigate the possible involvement of shell NAcc in the micturition control. Adult male Wistar rats (~450 g) with stainless steel guide cannulas implanted bilaterally in the shell NAcc 7 days prior to the experiments were anesthetized with 2% isoflurane in 100% O_2 and subjected to cannulation of the femoral artery and vein for mean arterial pressure (MAP) and heart rate recordings (HR) and infusion of drugs, respectively. The urinary bladder was cannulated for IP measurement. A miniaturized Doppler flow probe was placed around the left renal arterial for renal blood flow (RBF) recordings. After the baseline MAP, HR, IP and RBF recordings for 15 min, GABA (50 mM, 1 µL) or L-glutamate (50 mM, 1 µL) or saline (vehicle, 1 µL) injections were made bilaterally into the shell NAcc and the variables were measured for additional 30 min. Data are as mean ± SEM and submitted to Student's t test ($P < 0.05$). Bilateral injections of GABA into the shell NAcc (bilateral) significantly increased IP (168 ± 11% vs. 5 ± 3%, saline) and renal conductance (RC, 124.67 ± 23.51% vs. 5.45 ± 0.90%, saline), whilst a significant fall in MAP (–64 ± 2 mmHg vs. –2 ± 2 mmHg, saline) and HR (–92 ± 14 bpm vs. 1 ± 2 bpm, saline) were observed compared to saline injections. Bilateral injections of L-glutamate into the shell NAcc significantly increased IP (132 ± 18% vs. 5 ± 3%, saline), and MAP (13 ± 3 mmHg vs. –2 ± 2 mmHg, saline), whereas a significant decrease in RC (–7.39 ± 0.58% vs. 5.45 ± 0.90%, saline) and no changes in HR (13 ± 6 bpm vs. 1 ± 2 bpm, saline) were observed compared to saline injections. Conclusion: The shell NAcc participates in the neural circuitry involved in micturition control and plays a possible tonic role in the arterial pressure regulation.

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S2.P3

BIDIRECTIONAL EFFECT OF THE EXTREMELY LOW-FREQUENCY ELECTROMAGNETIC FIELD (50 HZ) ON BRAIN-DERIVED NEUROTROPHIC FACTOR LEVEL

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The impact of the extremely low-frequency electromagnetic field (ELF-EMF) on living organisms is still intensively and widely evaluated. Previous reports paid attention to its harmful effects such as sleep and mental disorders. However, ELF-EMFs are increasingly used in therapy of e.g. brain injuries. Some authors described ELF-EMF as a mild stress factor. Depending on the value of magnetic induction, the repeated ELF-EMF exposure 'turns on' different intracellular mechanisms: compensatory or deleterious ones. As the stress hormones (mainly corticosterone and noradrenaline) are known to modulate hippocampal function and may modify the plasticity processes in this area, we decided to determine the ELF-EMF-induced changes in BDNF (brain derived neurotrophic factor) level in hippocampus. Wistar rats were divided into three groups: control and exposed to 1 or 7 mT ELF-EMF. Rats were exposed to ELF-EMF 1 hour a day for 7 days. Control animals were subjected to the same experimental procedure as the exposed groups, except ELF-EMF exposure. The procedure was repeated three times with three week interval between exposures. After the end of each exposure the part of rats was sacrificed and brains were collected. The level of BDNF in hippocampus was determined. Our results showed that low-dose (1 mT) ELF-EMF increased the expression of BDNF, while the high dose (7 mT) ELF-MF reduced the expression of mRNA of the protein. Thus we concluded that the ELF-EMF effect on brain plasticity is bidirectional and depends on the value of magnetic induction of the field.

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S2.P4

EARLY-LIFE STRESS AFFECTS PERIPHERAL AND BRAIN RESPONSE TO IMMUNE CHALLENGE IN FEMALE RATS

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Early-life stress (ELS) is considered as a risk factor for mental and neurodegenerative disorders. Nowadays coexistence of blood-brain barrier (BBB) disturbances and inflammation appears to play an important role in the etiology, development and progression of those diseases. Data on females are still insufficient in this regard, so in our study, we focused on the consequences of ELS in female rats during preadolescence and adulthood periods. Specifically, we examined whether ELS, based on the maternal separation (MS) paradigm, can condition female subjects to other environmental factors later in life, such as an infection. To mimic this state, a single administration of bacterial lipopolysaccharide (LPS) was used. 24 h later, BBB permeability in the medial prefrontal cortex (mPFC) and hippocampus (Hp) was evaluated using fluorescent tracer and mRNA expression of tight junctions proteins (TJPs) and adhesion molecules representatives, *Cldn5*,*Ocld* and *Icam-1 was* measured, respectively. Moreover, serum levels of proinflammatory cytokines were studied and also their mRNA expression in the mPFC and Hp together with microglia markers (such as: *Aif1* and *Itgam*) and toll-like receptor 4 (*Tlr4*), representing the first line of defense against infections. Administration of LPS induced proinflammatory response on the periphery and in consequence increased BBB permeability, TJPs and *Icam-1* expression in the mPFC and Hp. Moreover, LPS enhanced mRNA expression of *Tlr4*, microglial markers and proinflammatory cytokines. Interestingly, the magnitude of LPS-induced effects was blunted in females previously subjected to MS. Within the studied brain regions, this was manifested mainly through suppressed mRNA expression of Icam-1, Tlr4, microglial markers and proinflammatory cytokines. However, those changes were more pronounced in adulthood than in preadolescence period. Particularly, tumor necrosis factor-α serum levels and the mRNA level of *Tlr4* and *Aif1* in the Hp were significantly lower in MS adult females. Concurrently, MS enhanced LPS-induced upregulation of *TJPs* expression in the Hp. These findings indicate that previous ELS experience may trigger adaptive response counteracting the impact of acute immune challenge in females.

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S2.P5

COMPARATIVE ANALYSIS OF THE INFLUENCE OF EPIPHYSIS AND SUPRACHIASMATIC NUCLEUS OF HYPOTHALAMUS ON VISION FUNCTION

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The main goal of the study was comparative analysis of the influence interaction suprachiasmatic nucleus of the hypothalamus and the pineal gland on the function of the structures of the visual analyzer. The experiments were carried out of rabbits. The electrophysiological parameters of the evoked potentials in the studied structures of the visual analyzer (retina, colliculus superior, visual cortex) were comparatively analyzed against the background of stimulation of the suprachiasmatic nucleus of the hypothalamus and pineal gland. On the basis of the obtained experimental results, it was effects influences of the suprachiasmatic nucleus and the pineal gland on the electrophysiological parameters of the evoked potentials of the structures of thge visual analyzer are opposite to each other.

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S2.P6

CONFIRMATION OF THE INFLUENCE OF AMIGDALA ON THE FUNCTIONS OF THE VISUAL ANALYZER STRUCTURES IN AMIGDALAR EPILEPSY

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As a result of the studies, it was found that the introduction of a solution of penicillin into the amygdala led to the development of prolonged convulsive activity. Ten minutes after the vision in elektroensofaloqramma shows epileptiform discharces. Epileptiform activity encompassed all structures of the brain. Such elektroensofaloqramma changes were recorded within 3–4 hours. The seizures reached a peak point within an hour, followed by a definite dynamics of epileptiform waves. Penicillin caused the appearance of generalized peaks in all structures of the visual analyzer. Sometimes were observed myoclonic seizures, rapidly developing into an epileptic seizure. elektroensofaloqramma analysis showed that epileptic activity first manifests itself in the amygdala, then in the visual cortex, the upper tubercles of the quadruple. Probably, the manifestation of such a sequence is associated with morphofunctional connections between these structures.

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S2.P7

THE ACTIVATED MICROGLIA IN HIPOCCAMPUS AS A CHARACTERISTIC OF STREPTOZOTOCIN-INDUCED MODEL OF ALZHEIMER DISEASE IN RATS

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Streptozotocin (STZ) induced model of Alzheimer disease (sAD) in rats is an equivalent of sporadic AD in humans. In the course of this disease appears many pathological features for example neurodegeneration of hippocampus. This pathology may be caused by neuroinflammation. In turn, it is effects activation of microglia. Wistar rats (N = 5) induced sporadic form of Alzheimer disease by intracerebroventricular (i.c.v.) microinjection of STZ (3 mg/kg) to check the level of microglia activation in this model 90 days after induction of sAD. The rats (N = 6) from control group (VEH) received a citrate buffer. Activation of microglia was checked using primary antibodies anti-macrophages/monocytes bound by secondary antibodies with fluorescent marker. Next using fluorescence microscope and a computer program Axio Vision photographed parts of hippocampus and counted number of activated cells with the use of calibrated frame. Analysis (Mann-Whitney test) of activation microglia in different parts of the hippocampus shows statistically significant differences in CA1 ($p < 0.01$; STZ $6.8 \pm 3.27/0.1$ mm^2; VEH $0 \pm 0/0$, 1 mm^2), CA2 ($p < 0.05$; STZ $4.8 \pm 1.92/0.1$ mm^2; VEH $0 \pm 0/0.1$ mm^2), CA3 ($p < 0.01$; STZ $4 \pm 1.73/0.1$ mm^2; VEH $0 \pm 0/0.1$ mm^2), DG ($p < 0.05$; STZ $7.25 \pm 3.6/0.1$ mm^2; VEH $0 \pm 0/0.1$ mm^2) parts of hippocampus. Activation of microglia was observed only in rats with sAD. All date are presented as mean ± SD. Conclusion: Activation of microglia in hippocampus only in animals with sporadic Alzheimer disease, suggest the increased neuroinflammatory response is presence even 3 months after induction of AD and may be the reason of behavioural disturbances.

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S2.P8

EFFECT OF SEROTONIN, ADRENALINE AND DOPAMINE ON THE FUNCTION OF THE VISUAL SYSTEM STRUCTURES

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It was revealed that the influence of the components of the monoaminergic system on the function of the structures of the visual analyzer is different. Thus, serotonin and dopamine have a positive effect on retinal function. However, the effect of dopamine was reflected in the 'a' wave of the electroretinogram, but did not affect the 'b' wave. The effect of adrenaline on the electrical activity of the investigated structures was negative. The effects of serotonin, adrenaline, and dopamine on electrical activity in the visual cortex similarly affect on the retina. All three components negatively influence the function of the superior colliculus.

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S2.P9

INFLUENCE OF CAFFEINE ON THE GENE EXPRESSION OF PROINFLAMMATORY CYTOKINES AND THEIR RECEPTORS IN THE HYPOTHALAMIC-PITUITARY UNIT

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Inflammatory cytokines are considered to be important mediators modulating neuroendocrine system at the level of the brain. The origin of these cytokines are differentiated. Some of them reach the brain parenchyma from periphery or are produced in the cells of choroid plexus; however these cytokines and their corresponding receptors are also expressed in the hypothalamic and pituitary cells both during homeostasis milieu and inflammation. Caffeine is one of the most widely consumed pharmacologically active substances which receptors are also widespread in the hypothalamic-pituitary unit. There are reports suggesting that caffeine may influence secretion of pituitary hormones however, this mechanism is not fully elucidated. The aim of the study was to determine the influence of caffeine on the expression of proinflammatory cytokines and their receptors in the hypothalamus and pituitary. The study was performed on sheep model. The experiment was carried out on 12 ewes intravenously injected with caffeine at the dose of 40 mg/kg (n = 6) or saline (n = 6). Animals were euthanized 3 hours after caffeine or saline injection. Hypothalamic tissue and anterior pituitary (AP) was dissected. The gene expression of cytokines such as: interleukin (IL)-1, IL-6 and tumor necrosis factor alpha (TNF-α) and their receptors IL-1R1, IL-1R2, IL-6R, gp-130, TNFR1, TNFR2 was determined. It was found that caffeine stimulated the gene expression of IL-6 both in the hypothalamus and pituitary, on the other hand caffeine suppressed the amount of TNF mRNA in these tissues. The effect of caffeine on IL-1β mRNA expression was differentiated, caffeine lowered the level of IL-1β in the AP, whereas it did not influence this mRNA level in the hypothalamus. Caffeine reduced the gene expression of TNFR1 and TNFR2 both in the hypothalamus and AP, but increased the amount of IL-1R2 mRNA in these tissues. Our study showed that caffeine exerted both stimulatory and suppressory effect on the gene expression of proinflammatory cytokines and some of these cytokines receptors in the hypothalamic-pituitary unit. This suggests that one of the mechanisms *via* caffeine influence the secretory activity of the hypothalamic-pituitary unit may be modulation of proinflammatory cytokines synthesis in this brain tissues.

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S2.P10

THE CB1 RECEPTOR ANTAGONIST REDUCES THE PRESSOR RESPONSE OF ANGIOTENSIN II AND ANGIOTENSIN 1-7 INJECTED INTO PARAVENTRICULAR NUCLEUS OF THE HYPOTHALAMUS IN CONSCIOUS NORMOTENSIVE RATS

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Previous experiments on anesthetized normotensive rats, have shown that intravenous injection of angiotensin II (Ang II) AT1 receptor antagonist losartan reverses the pressor response resulting from stimulation of cannabinoid CB_1 receptors (CB_1-R) in the paraventricular nucleus of the hypothalamus (PVN). The aim of this study was to determine the interaction between CB1-R and Ang II as well as Ang 1-7 in the regulation of blood pressure in conscious rats with spontaneous hypertension (SHR) and their normotensive control - Wistar Kyoto (WKY). All compounds were administered into the PVN through a stainless steel cannula and blood pressure was measured using noninvasive tail-cuff method (for a verification of normo- and hypertensive rats) and from the carotid artery (main experiments). Basal systolic (SBP), diastolic (DBP) and mean (MAP) pressure were approximately 60% lower in WKY compared to SHR. Angiotensin II (0.3 nmol/rat) and angiotensin 1-7 (0.3 nmol/rat) increased blood pressure both in WKY and SHR. The pressor effects of both compounds were stronger in SHR than in WKY. The increases in blood pressure stimulated by Ang II were inhibited by the antagonists of AT1 and AT2 receptors (losartan (8 nmol/rat) and PD123319 (10 nmol/rat), respectively) and those induced by Ang 1-7 were reduced by A779 (3 nmol/rat; the antagonist of Mas receptors) both in WKY and SHR. The CB_1-R antagonist AM251 (0.03 μmol/rat) significantly inhibited the effect of Ang II, and reversed the pressure response of Ang 1-7 in SHR and WKY. None of the solvents (DMSO, NaCl) nor antagonists caused a significant effect on blood pressure on its own. In conclusion, the CB_1 receptors in the PVN reduce the pressor response of Ang II and Ang 1-7 (elicited by the activation of AT1, AT2 and Mas receptors, respectively) in conscious normotensive and hypertensive rats.

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S2.P11

THE EFFECT OF NIACIN, VITAMIN B3, ON THE BETA-AMYLOID-ASSOCIATED PROCESS OF NEURODEGENERATION

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Alzheimer's disease (AD) is the most common form of dementia. Mechanisms of synaptic damage in AD are related to the neurotoxic effects of soluble forms of β-amyloid oligomers (AβO). AβOs attach to the synapses, inhibit synaptic plasticity, damage synaptic cytoskeletal proteins, and ultimately leads to synapse loss. The current direction of research is focused on identifying factors that protect neurons from the toxic effects of AβO. Because niacin is associated with energy metabolism, mitochondrial function, cell death, and aging, it could affect Aβ-dependent neurodegeneration. We aimed to assess the effect of two different forms of niacin: nicotinic acid (NA) and nicotinamide (N) on the Aβ-dependent neurodegeneration process. The second aim was to investigate whether NA and N could stimulate the neuroprotective effects of astrocytes. Experiments were performed on human cell lines: SH-SY5Y (neuroblastoma) and NHA astrocytes. The SH-SY5Y cells were differentiated for 10 days. 24 hours before the end of the experiment, the SH-SY5Y cells were incubated with CM (conditioned medium) derived from NHA astrocyte cultures: the control (CM), and treated with NA (CM-NA) or N (CM-N). After 1 h preincubation with CM, CM-NA or CM-N differentiated SH-SY5Y cells were incubated for another 24 h with Aβ (5 μM). The MTT test was used to assess the cytotoxicity of the tested factors. The expression level of *PSD95* (postsynaptic density protein 95, which plays an important role in synaptic plasticity) mRNA in neurons was assessed by qRT-PCR. Microscopic observation of autophagolysosomes (acidic vesicular organelles, AVO) in neurons was also performed. 24 hours incubation with Aβ decreased the viability of SH-SY5Y cells ($p < 0.001$). Incubation of SH-SY5Y cells with CM reduced the cytotoxicity of Aβ relative to control ($p < 0.02$). This effect increased significantly after the addition of CM-NA or CM-N ($p < 0.05$). We also observed that Aβ decreased the expression level of *PSD95* mRNA ($p < 0.01$) compared to the control, thus reducing the synaptic activity of differentiated SH-SY5Y cells. Concomitant use of Aβ with NA abrogated the synaptotoxicity of Aβ ($p < 0.01$). Microscopic examination showed that AVO formation was increased in Aβ treated SH-SY5Y cells ($p < 0.01$). AVO formation was antagonized by adding CM-NA CM-N ($p < 0.05$). CM from astrocyte cultures protects neurons from the toxic effects of Aβ. The use of two different forms of niacin: CM-NA or CM-N enhances this effect.

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S2.P12

SWIM TRAINING AMELIORATES OXIDATIVE STRESS IN THE SPINAL CORD OF AMYOTROPHIC LATERAL SCLEROSIS MICE

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Amyotrophic lateral sclerosis (ALS) is an incurable, neurodegenerative disease causing muscle atrophy. In some cases, ALS causes behavioral disturbances and cognitive dysfunction. Swimming has revealed a neuroprotective influence on the motor neurons in the SOD1-G93A mice model of ALS. In the present study, transgenic male mice overexpressing human SOD1 with G93A substitution, with wild-type B6SJL mice as controls were used. ALS mice were analyzed before ALS onset (10th week of life), at ALS 1 onset (first symptoms of the disease, ALS 1 onset and ALS 1 onset SWIM), and at terminal ALS (last stage of the disease, ALS TER, and ALS TER SWIM), and compared with wild-type mice. Swim training was applied 5 times per week for 30 minutes. The spinal cord was analyzed for the enzymes activities and oxidative stress markers. The present study identified the metabolic changes in the spinal cord already at the pre-symptomatic stage of the disease with the shift towards glycolytic processes at the terminal stage of ALS. Moreover, in the current study, we recognized the pathophysiological alteration resulting in a higher glutathione peroxidase activity in the terminal stage of ALS after swim training. Only slight modifications of oxidative stress markers were found. Nevertheless, they favor swim training as the protection against oxidative stress. Our results are relevant for therapeutic aquatic activity in ALS patients where physical activity recommendations still remain controversial.

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S2.P13

THE EFFECT OF BENZO[A]PYRENE ON OXIDATIVE STRESS IN CHICKEN EMBRYOS BRAIN

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Benzo[a]pyrene belongs to a large group of polycyclic aromatic hydrocarbons (PAHs). It is one of the most dangerous to health components of tobacco smoke with proven carcinogenic activity. Benzo[a]pyrene is also an important air pollutant and some food pollutants. The aim of the study was to show whether early exposure to benzo[a]pyrene affects the antioxidant system in the brain during the embryonic development of the bird. For this purpose, 100 fertile eggs of Ross 308 broiler parent stock were used. The eggs were divided into 5 groups as control, vehicle control, 0.1, 0.5, and 1 mg BaP/kg of egg weight. The eggs were injected to the yolk on day 6 of incubation. At day 14 of incubation, eggs were opened until 6 living embryos were obtained from each group. The activity of catalase, glutathione peroxidase (GPx), superoxide dismutase (SOD) and the level of glutathione (GSH) and malondialdehyde (MDA) were determined in the collected tissues. It was observed that, in benzo[a]pyrene-treated groups, the activity of SOD and GPx was increased. It was also indicated that the level of GSH was significantly decreased and the level of MDA - significantly increased. The greatest changes in the examined parameters were observed in the group of eggs injected with the dose of 1 mg BaP/kg of egg weight. These results indicate that *in ovo* administration of benzo[a]pyrene causes oxidative stress in the brains of chicken embryos.

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S2.P14

NEUROPHYSIOLOGICAL STUDY OF DISORDER AND RECOVERY OF SPATIAL MEMORY IN AN EXPERIMENTAL MODEL OF ALZHEIMER'S DISEASE

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The cluster the amygdala - the visual system - the olfactory analyzer normally jointly carry out the formation of behavior and spatial memory in the Morris water maze. Impaired memory and cognitive functions during the creation of an analogue of Alzheimer's disease in old albino rats was observed after bilateral surgical bulbectomy. Administration of curcuma solution after total memory loss in animals was accompanied by restoration of spatial memory. This was evidenced by the reduction in the latent time for searching for an invisible platform. The result of the effect of curcuma is the expression of neurotrophic factor and neurogenesis in the amygdala. This phenomenon can be explained by the effect of curcumins (active substances in curcuma) as a trigger for neurogenesis in the basolateral amygdala. This phase leads to the rehabilitation of cognitive function in the spatial disturbance of the connection between the amygdala and the olfactory receptor was possibly accompanied by structural rearrangements and increased interconnection and the development of new contacts between the amygdala and the visual system. The amygdala contains glutamatergic pyramidal neurons and GABA-ergic interneurons It became known that the amygdala, like the hippocampus and the bulb, is capable of neurogenesis in adult animals.

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S2.P15

EFFECT OF DIMETHYL FUMARATE ON DISORDERS OF THE OLFACTORY BULB NEUROGENESIS IN THE STREPTOZOTOCIN-INDUCED RAT MODEL OF ALZHEIMER'S DISEASE

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The sporadic late-onset form of Alzheimer's disease (sAD) affects 90% of AD patients and it is associated mainly with environmental factors. The intracerebroventricular injection of streptozotocin (STZ-icv) rat model of the sAD is widely used in basic research for understanding of the sAD pathophysiology and testing new therapeutic methods. One of the mechanisms contributing to memory impairment in AD is disruption of the postnatal neurogenesis in both the hippocampus and the olfactory bulb (OB). New neurons in OB determine the proper functioning of perceptual and memory processes related to smell, which in adults may be associated with adaptive mechanisms in response to environmental changes. It has been shown that patients with mild cognitive impairment and olfactory deficits are at higher risk of developing AD. Methods: young (n = 20) and old (n = 20) male Wistar rats were randomly divided into four groups (in each young n = 5 and old n = 5): 1) STZ+DMF with STZ-icv injection (3 mg/kg) and fed with chow containing DMF (0.4% by weight), 2) STZ with STZ-icv injection and fed with standard chow, 3) Sham+DMF with icv injection of vehicle (citrate buffer) and fed with DMF chow, 4) Sham – vehicle-icv and fed with standard chow. One week after STZ or vehicle icv rats were subjected to intraperitoneal injection of 5-bromo-2'deoksyurydine (BrdU) (50 mg/kg, once daily for three days) - a marker of cells undergoing cell division. Rats were sacrificed 9 days after the third BrdU injection and brain were subjected to immunofluorescent BrdU and doublecortin (DCX, marker of immature neurons) labelling. Data were analyzed using three-way ANOVA and Tukey's post-hoc test. Results and conclusion: both young and aged STZ rats were characterized by the lowest number of new immature neurons (BrdU+DCX containing cells) in the olfactory bulb ($p < 0.001$; STZ vs. other groups). STZ+DMF young and aged rats showed higher density of newly born neurons in the OB ($p < 0.001$) compared to STZ group. The highest percentage of immature neurons was observed in the OB of Sham+DMF rats in both age groups compared to the other experimental groups. STZ-icv causes significant disorders of neurogenesis in the OB. DMF therapy improved the disruption of adult neurogenesis in the OB induced by STZ-icv treatment.

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S2.P16

EFFECT OF PREBIOTICS SUPPLEMENTATION ON SOCIAL BEHAVIOUR AND PLASMA TNF-ALPHA LEVEL DISTURBANCES IN HIGH- AND LOW-RESPONDERS RATS WITH CENTRAL AMYGDALA HYPERACTIVATION

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Amygdala (Amg) hyperactivity as well as higher TNF-α plasma concentration occurs in patients with PTSD, anxiety disorders or depression. Long-term electrical stimulation (ES) induces abnormal hyperactivation of the Amg and can be used as an animal model of mentioned disorders. The central nucleus of the Amg (CeA) contribute to neural and endocrine responses to stress and it's functioning is associated with anxiety and social behaviour. Moreover Amg is considered to have a crucial role in the microbiota signals processing and integration. Between the gut microbiota and central nervous system exists a functional bidirectional communication. Manipulating the gut microbiome may improve host mental health and reduce inflammatory processes. Galactooligosaccharides (GOS) are prebiotics - non-digestable polysaccharides that increase the growth and activity of health-promoting microorganisms. Individual differences in the neuronal, endocrine and immune responses to stress may result in different susceptibility to the development of anxiety disorders in humans. The rat model in which male rats are divided into high responders (HRs) and low responders (LRs) - based on their locomotor activity during exposure to the novel environment - is well-described and reflects differences observed in humans. Twenty eight male Wistar rats categorized as HRs or LRs in the novelty test were subjected to 14-day electrical stimulation of the CeA and 21-day supplementation with GOS. Three chamber sociability test was used to assess social behaviour. One hour after the test blood samples were collected and centrifuged to obtain plasma. TNF-α plasma concentration was determined by ELISA test. Data were analyzed using three-way ANOVA and Tukey's post-hoc test. The long-term ES of the CeA caused deficits in social behaviour (avoiding interaction with a non-familiar rat and spending more time with the familiar rat; $p < 0.01$). It also led to a significant increase in plasma TNF-α level ($p < 0.001$). In HRs rats these effects were enhanced. GOS supplementation improved disturbances induced by the ES of the CeA as stimulated and supplemented rats exhibited more prosocial behaviour and lower TNF-α plasma concentration ($p < 0.05$). GOS therapeutic effects were more pronounced in LRs rats.

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S2.P17

IMPROVED MOTOR FUNCTION AS A RESULT OF THE INFLUENCE OF MINOCYCLINE ON MOTOR CORTEX NEURONS IN CORTICAL MODEL OF PHOTOTHROMBOTIC ISCHEMIC STROKE IN RATS

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Cerebrovascular diseases are the principal causes of mortality and disability worldwide. In survivors, strokes can result in long-term disability. Ischemic strokes constantly pose for great amount of mortality worldwide. Minocycline, by launching plethora of neuroprotective mechanisms may be beneficial as the treatment which has been confirmed in many research models of acute brain damage. Therefore, it is important to search for neuroprotection mechanisms that would allow to extend the therapeutic window and develop new strategies for treating ischemic strokes. The first goal in our research was to develop experimental models of cerebral ischemia that mimic human stroke. The second aim was to investigate the effect of minocycline on penumbra and functional outcomes after ischemic stroke. Photothrombotic ischemia of motor cortex was produced in 72 male Long-Evans rats. We tested different time windows: 24 h, 48 h and 7 days after stroke induction. Half of the experimental groups received an intravenous dose of minocycline (1 mg/1 kg b.w/1ml solution, 10 minutes after stroke). CatWalk XT, Grip Strength-test and elevated runway-tests were performed. These functional tests were applied before and after ischemic stroke. In groups with minocycline we observed statistically significant improvement of speed of walking, correctness of the stepping pattern and increase of grip strength. Penumbra and necrosis were localized by immunohistochemical techniques and we measured its size and quality. Conclusion: minocycline improves motor function in ischemic rats. Minocycline action also correlates with size of necrosis and penumbra but determining the exact relationship between them requires in-depth studies.

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S2.P18

CHANGES IN THE RESPONSIVENESS OF THE RAT DORSOMEDIAL HYPOTHALAMUS TO DIFFERENT METABOLIC CONDITIONS UNDER HIGH-FAT DIET

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Food intake and metabolism are controlled by a network of brain structures, most of which are located within the hypothalamus. Feeding behaviour undergoes regulation by homeostatic signals incoming from the digestive system (*via* the hunger and satiety signalling peptides), as well as by the circadian clock, driving an increased food intake during the active, and reduced during the behaviourally quiescent phase. Therefore most animals display a natural preference or restriction of feeding to a particular part of the day. The brain structure especially responsive to restricted feeding is the dorsomedial hypothalamus (DMH). In this study we aimed at verifying how the DMH responds to different metabolic states in both *ad libitum* and restricted-fed rats. Moreover, we checked whether these responses change under 4-week-long high-fat diet (HFD). For this we performed immunohistochemical staining for the cFOS protein, an early response gene reflecting changes in the neuronal activity. In the first protocol, rats which had been fed *ad libitum*, were then food deprived for 48 h (hunger). Following, one group was refed for 2 h (full satiety). In the restricted-feeding (RF) protocol, rats had a limited access to food to 6h every day during the night (active phase) for a period of two weeks. Separate groups of animals were then culled either 0.5 h before, or 1.5 h and 3.5 h after the scheduled meal. The experiment revealed that in both protocols and for both diets, the number of cFOS positive cells in the DMH is the lowest during hunger and the highest after feeding. However, HFD-fed animals showed a higher increase in cFOS immunoreactivity after a refeed following food deprivation. On the other hand, in HFD restricted-fed animals, the increase in cFOS after a scheduled meal was lower than in the control group. These results highlight the involvement of DMH in the processing of the information about the metabolic states of an organism and present ways in which HFD disrupts its responsiveness to satiety, which differ depending on the feeding schedule.

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S2.P19

MODULATION OF PAIN IN BRAIN LIMBIC AREAS: ROLE OF OPIOID AND CANNABINOID SYSTEMS

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The development of pain as a common experience and its treatment is very important, not only where it is caused by injury or inflammation, but also in chronic states where the nerves themselves are damaged. Non-steroidal anti-inflammatory drugs (NSAIDs) are the most widely used analgesics. However, a few recent studies have demonstrated that these non-opioid drugs in the case of their prolonged use, elicit the opioid-like effect, tolerance may entail serious adverse effects. The brain limbic system is involved in affective-emotional aspects of pain and this study has shown brain mechanisms of non-opioid induced antinociceptive tolerance to NSAIDs in the 'formalin test'. Opioids remain the drug of choice for the clinical management of moderate to severe pain and play a large role in the pain modulatory system. Studies over the past decade have shed light on the influence of endocannabinoids on the opioid system. Evidence from both animal and clinical researches point toward an interaction between these two opioid and cannabinoid systems, and suggest that targeting the endocannabinoid system may provide novel interventions for managing morphine addiction, opiate dependence and tolerance, and of withdrawal reactions. In this study, we present new experimental data indicating that microinjections of widely used non-opioid, in particular, NSAIDs diclofenac, ketoprofen, ketorolac, and lornoxicam into pain matrix key structures of brain limbic areas, such as the rostral part of the anterior cingulate cortex, agranular insular cortex and central nucleus of amygdala (CeA) of rats, - induce antinociception. When administered repeatedly, tolerance developed to the antinociceptive effects of these drugs. Pre- or post-treatment with opioid receptor antagonists, naloxone and CTOP as well as cannabinoid CB1 receptor antagonist AM-251, separately or in combination in the CeA, prevented or abolished antinociceptive effects of these non-opioid analgesics. These new findings confirmed the concept that antinociception and the development of tolerance to NSAIDs are mediated *via* endogenous opioid and cannabinoid systems involving the descending pain modulatory circuits attenuating pain behavior in rats - defensive withdrawal reflexes at the spinal cord level. The crucial structures of this descending pain modulatory system are midbrain periaqueductal grey matter and rostral ventro-medial medulla. These findings, thus, emphasized the important role of these limbic regions, the rostral anterior cingulate cortex, agranular insular cortex, and central amygdala in rats' pain behavior.

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S2.P20

MINOCYCLINE AFFECTS SPLEEN T AND B LYMPHOCYTES PERCENTAGE IN STREPTOZOTOCIN-INDUCED MODEL OF ALZHEIMER'S DISEASE IN RATS

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Minocycline was shown to have anti-inflammatory and neuroprotective effect in neurodegenerative diseases. Here, we investigated immunomodulatory properties of minocycline (MINO) at a dose of 35 mg/kg b.w., administered intraperitoneally (i.p.) for 7 consecutive days, on immune system in rats with STZ-induced model of sporadic form of Alzheimer's disease (sAD). Thirty male Wistar rats were divided into groups: STZMINO (intracerebroventricular, i.c.v., streptozotocine (STZ) and i.p MINO injections), VEHMINO (i.c.v. vehicle and i.p minocycline injections), STZ (i.c.v. STZ injection), VEH (i.c.v. vehicle injection). Spleens were collected and homogenized. Flow cytometry with a three-color combination of fluorescent monoclonal antibodies was used to identify T (CD3+), B (CD45RA+), NK (CD161a+) lymphocytes and T lymphocyte subsets of CD4+ and CD8+ lymphocyte percentages in spleen supernatants, according to the method that we previously described. Statistical significance was ascertained by U Mann-Whitney test and the results were considered significant at $p \leq 0.05$. Data is presented as mean percentage (%) of cells ± SD. As a result significantly ($p < 0.05$) increased T lymphocytes (STZMINO: 40.82 ± 9.61; VEHMINO: 42.13 ± 3.71) and B lymphocytes (STZMINO: 17.05 ± 2.64; VEHMINO: 24.2 ± 8.29) percentages were observed in minocycline-treated groups relative to corresponding T lymphocytes (STZ: 20.4 ± 1.16, VEH: 17.75 ± 3.65) and B lymphocytes (STZ: 7.35 ± 1.71, VEH: 11.87 ± 1.97) percentages in non-treated animal groups. Moreover, percentages of T lymphocytes subsets TCD4+ (39.5 ± 7.18) and TCD8+ (16.09 ± 3.16) in STZMINO group were significantly ($p < 0.05$) increased as compared to corresponding STZ (TCD4+: 15.58 ± 3.4; TCD8+: 9.09 ± 1.45) groups. The results indicate that minocycline changes spleen lymphocyte distribution, including $TCD4^{+}/TCD8^{+}$cell ratio in the sAD model.

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S2.P21

EFFECT OF KETOGENIC DIET ON NEURODEVELOPMENTAL REFLEXES

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The ketogenic diet is a type of nutritional system based on deriving energy from fats while minimising carbohydrate intake while maintaining an adequate amount of protein in the diet. It is currently used for the treatment of drug-resistant epilepsy with very good results. There are many studies showing the positive effect of diet on the nervous system in the case of neurodegenerative disorders, endocrine immune disorders, obesity, diabetes and certain types of cancer, while there is a lack of data on the effect of diet on the development of the nervous system. It is important from the point of view of the use of diet by pregnant women. In order to test the effect of diet on neurodevelopment, a series of experiments were carried out on Wistar rats. These tests aimed to check reflexes such as forelimb grasp, hindlimb grasp, righting, hindlimb placing, cliff avoidance, gait, auditory startle, posture, eye openings and accelerated righting. The rats were divided into three study groups: a control group on a normal diet (ND) , rats on a prenatal ketogenic diet (KD/ND) and a group on a ketogenic diet until P21. The first conclusion is a significant difference in weight and weight gain between the groups and in body hair. Rats on ketosis are smaller and less hairy. My research shows that there is a trend when pregnant female Wistar rats are on a diet that indicates a difference in reflexes often to the disadvantage of rats on a ketogenic diet. The longer a group was on the ketogenic diet the later they developed reflexes.

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S2.P22

ARCHITECTURE OF A FUNCTIONAL SYSTEM OF THE SAGITTAL BALANCE MAINTAINING

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Body balance is required for a static position and locomotor function. Its maintaining means alignment of the different levels structures of the body in space with respect to each other for organizing the skeleton geometry and locomotor apparatus structure so as to ensure their correspondence to gravity. In the form of mechanical stress this environmental factor affects the processes of adaptive remodeling of bone tissue, and a number of other structures with it. The state of the bone tissue is even considered as a reflection of the effects of gravity on the body in the evolution. The balance of the body in space can be characterized by such an integrative indicator as the sagittal balance of the spine - the vertical alignment of the trunk above the pelvis. Its quantitative characteristic is the horizontal distance between the centers of the body of the 7th cervical vertebra (C7) and the posterosuperior border of the sacrum on lateral radiographs of the spine in full growth. To understand the mechanisms of maintaining the sagittal balance within physiological borders, it is important to consider it within the P.K. Anokhin's concept of functional system. In this case, the sagittal balance should take a central place forming the functional system, being the final adaptive result of functioning, that is, one of the parameters of homeostasis, for the maintenance of which all functional systems of the body are formed. In the architecture of such a functional system, it is necessary to distinguish functional blocks - structures that interact with each other to maintain this parameter and perform a certain role, for further multilateral study of such systems. The receptors that determine homeostasis parameter (sagittal balance) deviations and trigger the functional system are vestibuloreceptors, proprioceptors, and the visual sensory system. In the physiological center, which includes a number of central nervous system structures, the resulting afferentation is integrated in the creation of motor programs. At each current moment, there are changes in the state of the body's executive structures involved in maintaining the sagittal balance. Such efferent structures are not only skeletal and muscle macrostructures, but also elements of bone microarchitecture. They undergo changes in a shorter time interval during remodeling, ensuring the body adaptation with the external environment at higher levels of interaction. Consideration of the structures involved in maintaining the sagittal balance from this point of view will allow us to determine the possible causes of its displacement and methods of its correction.

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S2.P23

EFFECT OF PROCAINE BLOCKADE OF THE VENTRAL TEGMENTAL AREA ON THETA RHYTHM INDUCED BY PHARMACOLOGICAL ACTIVATION OF THE PEDUNCULOPONTINE NUCLEUS

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The ventral tegmental area (VTA) and the pedunculopontine nucleus (PPN) are structures that have important influence on the induction and regulation of hippocampal theta rhythm, which plays a key role in important processes such as memory and REM sleep. PPN is one of the initial structures of an extensive theta rhythm induction network, additionally - one of the nuclei that sends cholinergic projections to the VTA. Recent studies has also shown that VTA stimulation is accompanied by the theta rhythm in the hippocampus. However, the functional relationships between these structures and hippocampal theta rhythm is still not fully understood. The aim of the experiment was to investigate the effect of pharmacological cholinergic activation (carbachol) of PPN and inactivation (procaine) of VTA on the formation and regulation of hippocampal theta rhythm. The surgery was performed under urethane anesthesia (maintained at such a level that theta rhythm does not appear spontaneously). Rats were implanted with the use of stereotaxic frame with bilateral hippocampal recording electrodes and bilaterally with standard pedestal guides for infusions to the VTA and PPN. Local field potential (LFP) was recorded from the hippocampal electrodes during the whole experiment with the use of Spike-2 softwere. Total power in the hippocampal signal was analyzed. Theta and delta bands peak power (P_{max}) was extracted. P_{max} value in the theta frequency band (3–4 Hz and 4–5 Hz) temporarily decreased after intra-VTA injection of procaine during carbachol - induced theta episode in comparison to control group. P_{max} value in the delta frequency band (1–2 Hz and 2–3 Hz) temporarily increased after intra-VTA injection of procaine during carbachol-induced theta episode in comparison to control group. In control group - water injection to VTA during carbachol - induced theta rhythm episode had no effect in the signal power (P_{max}) in both theta and delta bands. The results suggest that the VTA probably might be a part of the broad network involved in theta rhythm induction.

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S2.P24

MET-ENKEPHALIN INVOLVEMENT IN THE PROTECTION OF CEREBELLAR AND FRONTAL CORTEX IN VAGOTOMIZED RATS

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Met -enkephalin, one of the opioids peptide, exerts protective effects on the pain sensation, mood, modulation of all endocrines axis and dopamine activity in the brain. Dopamine and other monoamines are involved in depressive disorders in parkinsonians and epileptic patients. Moreover, evidences were found, that vagus nerve which is connected with the gastrointestinal system, is affected very early in the parkinson disease. Recently, it was also postulated that opioids and cholinergic systems interacts (*via* vagus nerve) at the level of brain - gastrointestinal axis. The question arises about the role of opioids in the regulation of vagus nerve effect on the brain structures connected with movement and mental disorders. Thus, the aim of this study was to examine the effect of vagotomy on the activity of Met-enkephalin in the cerebellar cortex (CBR) and frontal cortex (Fctx) in rats during control and inflammation conditions. Experiment was performed on adult, male Wistar rats divided into seven groups (8 rats/group): 1) control without treatment (CNT); 2) NaCl (injection of NaCl); 3) Sham surgery and injection with NaCl (Sham); 4) Subdiaphragmatic vagotomy (Vgax); 5) NaCl treated with lipopolysaccharide (LPS) (NaCl+LPS); 6) Sham +LPS; 7) Vags+LPS. Following surgeries, animals recovered for 30 days, then were injected i.p. with NaCl or LPS (10 μg/rat *E.coli* 026:B6). Two hours later, animals were euthanized and cerebellar cortex and frontal cortex were isolated, weighed and stored at –80°C. Native Met- enkephalin concentration in the brain structures was estimated by radioimmunoassay method. Vagotomy increased the level of Met-enkephalin in the CBR by 86% ($P < 0.01$), LPS also caused higher level of opioid (by 68%, $P < 0.05$). Unexpectedly, LPS did not potentiated the vagotomy effect on Met-enkephalin concentration (increase only by 14%). Fctx Met-enkephalin level was increased by 4.4 times compared to CNT group; LPS caused increase of opioid by 140%. In contrast to CBR, LPS potentiated the opioid response to vagotomy and induced 335% increase compared to CNT group. In conclusions: 1) CBR and Fctx opioid system similarly responded to vagotomy (increase) but showed different sensitivity to inflammation; 2) Met-enkephalin protective effect on cholinergic system under inflammation condition in vagotomized rats was significantly higher in frontal cortex than in cerebellar cortex.

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S2.P25

FREQUENCY-DEPENDENT PLASTICITY OF SPONTANEOUS ACTION POTENTIALS WITHIN IDENTIFIED *LYMNAEA'S* NEURONS

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Action potential (AP or spike) is a key reaction of neuronal membrane underlying the responses of the nervous cells on any type of irritation. Some neurons are able to generate spontaneous AP independently from external impact (pacemaker neurons and pacemaker potentials). There is no doubt that AP duration can determine the total excitability of the neuronal membrane due to ability to regulate a time course of it refractory period, i.e. defines a firing rate of the neurons which is critically important for correct operation of neuronal networks. In our days, an initial point of view on AP as 'all-or-none', permanent-type event is considerably revised. The plastic nature of AP has been described in various neuronal types both in vertebrates and invertebrates. As a rule it is talking about frequency-dependent broadening of neuron soma spikes, evoked by intracellular current injection. From the other hand, AP variability during spontaneous activity is less described. We report about frequency-dependent changes in the duration of various AP phases: depolarization (DP), repolarization (RP) and undershoot (US) in identified central neurons of mollusk *Lymnaea stagnalis*. Glass microelectrodes were used for a 10 min record both for giant dopamine- (RPeD1, $n = 6$) and serotonin- (LPeD1, $n = 4$) containing cells in isolated CNS preparations. Based on initial data distribution of frequencies, all spikes in a record were combine in 3 groups - low (first quartile or less), base (between first and third quartiles), high (third quartile or more) and then analyzed by conventional electrophysiological (InputWin) and statistical (Statistica 6.0) tools. For RPeD1 we were able to observe a slight AP broadening both for DP and RP phases at high frequencies, while for LPeD1 about two-time decrease of duration in all AP phases under the study was determined, meaning compression of the signal. AP amplitude is also varied in frequency-dependent manner in RPeD1 and LPeD1 - both a positive (an area above) and a negative (an area below rest potential value) phases of AP decline with spike frequency rise. However, total time of neuronal membrane depolarization and hyperpolarization state was significantly increased at high frequencies in both cells. We hypothesize that mentioned above differences in AP frequency-dependent plasticity can underlie variations in the efficiency of interneuronic communication in dopamine- and serotoninergic networks of molluscan brain.

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