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Corrections to the abstracts of the 8th Polish Neuroscience Society Congress

P6.04 Induced glia activation is harmful for neurogenesis of human umbilical cord blood neural stem cells

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Brain inflammation contributes to the propagation of the neuropathological events that involves activation of astrocytes and microglia. It remains obscure how activated glial cells affect the survival and differentiation of NSC. The aim of the study was to analyse neuronal commitment of HUCB-NSC cultured in the presence of normal and LPS or TMT activated glial cells. Labeled HUCB-NSC were seeded on confluent monolayer of normal or stimulated astrocytes and microglial cells isolated from neonatal rat brain and cultured for 7DIV. Normal rat astrocytes induce HUCB-NSC to differentiate mostly into neurones (75% TUJ1+; 65% MAP-2+), microglia stimulate HUCB-NSC to differentiate into neurons (45% TUJ1+) as well as into astrocytes (56% S100B+). Induced astrocytes diminish neurogenesis of HUCB-NSC (29% and 33%, respectively, vs. 75% TUJ1+) and increase astrocyte differentiation (52% and 53%, respectively vs. 1% S100B+) comparing to non-stimulated astrocytes. Microglia activation decreases HUCB-NSC differentiation into neurons (27% and 26% respectively vs. 45% TUJ1+) but enhances oligodendrogenesis (9% and 7% respectively vs. 1% O4+) compared to normal microglia. Activation of microglia and astrocytes induced by LPS and TMT attenuate proneural effect of non-stimulated (resting) glia co-culture. Interaction with glial cells modified by inflammation is crucial for NSC survival and differentiation after brain insult. Supported by grants: 2P05/A177/29; 1309/P01/2006/31.

P12.01 Functional and structural changes in rat medial gastrocnemius muscle after spinal cord transection

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In adult rats after a complete spinal cord transection (SCT) at low thoracic level, the locomotor movements of hindlimbs are impaired. Due to reduced activity, the structural and contractile properties of hindlimb muscles are considerably changed. Previous investigations of effects of the SCT on two antagonistic muscles: fast dorsi flexor (extensor digitorum longus; EDL) or slow extensor (soleus; Sol) demonstrated that contractile properties of both muscles tended to become similar. To verify whether the way of muscle transformation depends on the muscle physiological function or on the primary muscle fiber composition, we investigated rat medial gastrocnemius muscle (fast extensor) that is composed of three different types of motor units. In our study, the changes in EMG activity, contractile properties and the contents of myosin heavy chain (MHC) isoforms in adult rats were studied one, three and six months after SCT at low thoracic level. We demonstrated that just one month of reduced muscle activity evoked by SCT resulted in the significant changes in muscle contractile properties that were maintained later on. The fast gastrocnemius muscle became slower and weaker (as EDL m.) but surprisingly at the same time it became more fatigable and composed of fast MHC isoforms only (as Sol m.).

P12.07 Spinal cord transection alters NGF and its p75 receptor expression: Does exercise counteract it?

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The p75 receptor (p75NTR) binds growth-promoting neurotrophins (NTs) as well as Nogo receptor for growth-inhibiting proteins, so it is well situated to gauge the balance of positive and negative influences on axonal plasticity. In the adult rat spinal cord p75NTR expression is limited to nerve growth factor (NGF)-responsive DRG primary fibres, but motoneurons disclose potential to re-express p75NTR following nerve injury. Since altered p75NTR may affect remodeling of spinal network, here we examined whether complete spinal transection at Th 9 segment (1) changes p75NTR in DRG central processes (2) induces NGF/ p75NTR in spinal cells rostrally and caudally to injury. NGF immunoreactivity (IR), undetectable in intact rats, was induced in dorsal horn neuropil at 3 and 10 days after transection. By 6 weeks it tended to decrease whereas p75NTR IR fibers distributed in laminae I–IV and upholstering dorsal funiculus, maintained IR rostrally and caudally to injury. In its proximity their appearance was distorted and p75NTR distribution altered. An induction of IR was found in glia in lateral and ventral funiculi. As long-term locomotor exercise is a potent cue to stimulate spinal NTs and locomotor recovery in spinalized animals, we evaluated in parallel its effect on NGF/p75NTR expression. Training increased p75NTR protein in intact rats by 12%, but did not cause gross change in p75NTR IR pattern in either group of rats. Results suggest lack of modulatory potency of training on NGF/p75NTR signaling following spinalization. Supported by SPN- 007/P07 Polish-German MSE/BMBF grant.

P14.15 Neuroprotective effects of methylnicotinamide in cultured cerebellar granule cells

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The aim of this study was to evaluate neurotoxic and/or neuroprotective effects of 1-methylnicotinamide in primary cultures of rat cerebellar granule cells. It has been suggested that MNA, may be neurotoxic due to mechanism resembling that of MPP+, while neuroprotective effects of its parent substance nicotinamide (NAM) has been demonstrated. Excitotoxicity was induced by 30 min incubation with 0.5 mM glutamate or 0.5 mM NMDA + 5 μM glycine. Neurotoxicity was evaluated 24 h later with propidium iodide staining. Our initial studies demonstrated very weak neurotoxic potential of both MNA and NAM, which in concentrations below 50 mM were devoid of neurotoxicity during 24 h cell exposure, whereas 50 μM MPP+ induced a considerable neurotoxicity. Subsequent experiments demonstrated that 25 mM NAM and MNA significantly reduced NMDA-evoked neurotoxicity. They also suppressed glutamate- or NMDA-induced $45Ca^{2+}$ uptake in CGC and glutamate-evoked increases in the intracellular Ca^{2+} level detected with fluorescent probe Calcium OrangeTM. These results primarily suggested that both NMA and MNA might directly interfere with activity of NMDA receptors. However, further experiments showed that MNA and NAM do not interfere with NMDA + glycine evoked [3H]MK-801 binding to rat brain membranes, pointing to other mechanism of neuroprotection. In conclusion, our results demonstrate that MNA is not neurotoxic, but instead induces neuroprotection in the excitotoxic insults comparable to NAM. The mechanism of this phenomenon remains unclear. Supported by grant PBZ-KBN-101/T09/2003/11.

P16.01 Chronic stress and neurogenesis in structures of the limbic system in rat

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The limbic system is involved in response to stress stimulation. Incorporation of bromodeoxyuridine (BrdU) into DNA can serve as a marker of cells division associated with neurogenesis. In this study we investigated an influence of the chronic open field (OF) and forced swim (FS) stimulation on the level of cell proliferation in the hippocampus, amygdala and hypothalamus in the young and adult rats. 26 rats of ages P7 and P360 were exposed to chronic stress lasting 15 minutes daily for 21 days. BrdU was administered three times and brains were stained using immunohistochemical method. In the control groups BrdU-ir cells were observed in all examined structures, but their level was different. Moreover there were a large number of BrdU-immunoreactive cells in P7, whereas moderate in P360. In the rats exposed to chronic OF and FS the number of BrdU-ir cells in the investigated structures was considerably decreased in the young rats and slightly decreased in the adult ones in comparison to the controls. Little differences in the amount of BrdU-ir cells were observed after exposure to chronic stress both types. Our results indicate that newly proliferated cells are present in the investigated areas of the limbic system both in the young and the adult rats. Suppression of cell proliferation after chronic OF and FS is higher in the adult rats. Type of applied chronic stress does not have an influence on the neurogenesis level.

P16.03 Age-related changes of BDNF and TrkB distribution under chronic stress exposure in rat

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Chronic stress affects brain areas involved in learning and emotional responses. The brain-derived neurotrophic factor (BDNF), by acting through its receptor TrkB, is essential regulator of synaptic function, growth, and neuroprotective processes. BDNF activation is caused by both physical and psychological stress events. The present study examines the effect of chronic stress on BDNF and its receptor TrkB pattern in selected brain structures of the young (P7) and old (P360) rats. Twenty-six male Wistar rats were exposed to 15 min forced swimming daily during three weeks. Fluorescent immunohistochemistry was used to localize BDNF and TrkB in brain structures connected with stress response: paraventricular nucleus (PVN), supraoptic nucleus (SO), central and medial amygdaloid nuclei (CeA and MeA). Density of BDNF and TrkB positive cells in control animals is higher in P7 than P360. However, after chronic stress exposure an increase of these markers is considerably higher in the adult than in the young animals. In particular, we observed the increase of BDNF and TrkB in the magnocellular but not parvocellular part of PVN. In the Amygdala density of these markers was lower in MeA than in CeA. The results support the view that habituation to the stress conditions is easier in the early postnatal period.

P16.09 Stress-induced changes of interleukin-1beta (IL-1beta) within the limbic system in rat

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IL-1beta is the proinflammatory cytokine highly produced by CNS glia under conditions of disease, damage and stress. It is reported that cytokines are released not only in response to the traumatic stress, but also immobilization or psychophysical stress. Many studies suggest that acute stress increases the level of cytokines, but the reports on the chronic stress are contradictory. The aim of this study was to compare the influence of different chronic stress stimulations on IL-1beta level in relation to age. We applied psychological (open field) and psychophysical (forced swimming) chronic stress to investigate IL-1beta changes in hypothalamus, hippocampus and amygdala in the young (P7) and adult (P360) male Wistar rats. Activation of the limbic system under the chronic stress was estimated by c-Fos expression level. IL-1beta immunoreactivity was detected by immunohistochemical staining and semi-quantitative protein expression was estimated with chemiluminescent immunoblotting. We found no differences in c-Fos level in the limbic system in the young rats exposed to both types of chronic stress and the control groups. The adult rats, however, presented an increased c-Fos expression under both chronic stress conditions. Our results indicate the decreased expression of IL-1beta under the chronic stress exposures in both age groups compared to the controls. The highest expression of IL-1beta we observed in the hippocampus of P7 and P360, however, the protein level was lower in the adult rats.

Abstracts missing

E1 New transgenic mouse lines for temporally-controlled targeted somatic mutagenesis in astrocytes

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The functions of astrocytes, in particular their interactions with synaptic connections, are mainly derived from *in vitro* studies, but their relevance *in vivo* remains elusive, mainly due to a lack of animal models. We decided to establish transgenic mice that enable temporally controlled targeted mutagenesis in defined subpopulations of astrocytes based on the Cre-ERT2 system (Metzger and Chambon 2001). To drive expression of Cre-ERT2 in astrocytes, we generated lines carrying Cre-ERT2 under the control of large genomic DNA fragments of astrocyte-specific promoters (GLAST, Connexin-30, Aquaporin-4, ApoE) contained in bacterial artificial chromosomes (BACs). A first characterization of our transgenic lines in combination with reporter mice revealed that (a) Cre expression patterns across different organs and brain regions matched the activity pattern of the driving promoter, (b) transgenic lines derived from a given construct differed in the level of Cre-mediated recombination but not in its regional distribution and (c) Cre activity was strictly tamoxifen-dependent. Out of the four promoters chosen, Glast and Cx30 induced strong Cre expression in brain cells with non-overlapping regional distribution, whereas ApoE-CreERT2 and Aqp4-Cre-ERT2 lines showed low levels of Cre activity in the CNS. In GLAST-CreERT2 lines, Cre-mediated recombination occurred in cerebellar Bergmann glia, retinal Mueller cells, as well as in the olfactory bulb, cortex, hippocampal dentate gyrus and subventricular zone. In contrast, in Cx30-CreERT2 lines the highest level of recombination occurs in midbrain, thalamus, hypothalamus and brain stem. The initial characterization indicates that the new transgenic mouse models will help to determine the relevance of astrocytes during brain development and in the adult. Supported by DFG SPP1172, CNRS.

E2 Ischemia effects ECM-FAK signaling pathway in gerbil hippocampus

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Cell attachment with ECM proteins generates intracellular signals which lead to the specific tyrosine phosphorylation cascade of limited number of protein substrates, and these participate in the regulation of cytoskeletal organization and gene expression. This pathway involves FAK a non-receptor tyrosine kinase, a key component responsible for the flow of information from the ECM to the cell interior. Phosphorylated FAK may interact directly with other kinases, adaptor molecules and cytoskeletal proteins, perhaps providing a pathway by which ECM may regulate cell viability. Therefore it remains conceivable, that excessive matrix protein degradation in pathological conditions may lead to the disruption or even loss of the cell-ECM interaction and can further affect the downstream signaling pathways. This prompted us to verify if ischemia induced degradation of ECM proteins is temporally coincident with the modulation of intracellular pathway to which it is connected. We sought to clarify this point by measuring the activity of metalloproteinases – MMP-2 and MMP-9 as well as the activity of intracellular protein – FAK. We found that short-time forebrain ischemia leads to the activation of MMPs in CA1 region of the hippocampus at 48 and 72 h of reperfusion. At the same time a significant decrease of total FAK protein as well as its activation/phosphorylation, and the reduced amount of FAK/Src complex was observed. It may be concluded that ischemia-induced changes in ECM-FAK signaling pathway may contribute to delayed neuronal degeneration. Sponsored by MES grant 2P05A 09928.

Abstract “Expression of chemokine protein sin the rat model of temporal lobe epilepsy” by Lukasiuk K., Guzik A., Sliwa A. has been duplicated and occurred as P6.03 and P7.09. The abstract will be presented as P7.09.
Page 272, title of Symposium V, IS: Novel data processing algorhithms in clinical neurophysioology”, SHOULD BE: “Novel data processing algorithms in clinical neurophysiology”.

Misspellings in Authors' names

P3.11	IS: Tarnowska A., SHOULD BE: Sarnowska A.
P9.02	IS: Bginskas A., SHOULD BE: Baginskas A.
P10.18	IS: Sitarski T., SHOULD BE: Witarski T.
P10.18	IS: Nawrot D., SHOULD BE: Nawrat D.
P10.19	IS: Kamienska D., SHOULD BE: Kaminska D.
P14.17	IS: Kamienska B., SHOULD BE: Kaminska B.
P16.16	IS: Marin Ch., SHOULD BE: Mawrin Ch.

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Authors, Organizers of the Congress, and Editors of Acta Neurobiologiae Experimentalis apologize for these errors.