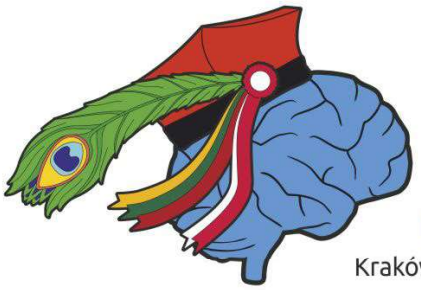


FENS
REGIONAL MEETING
Kraków, Poland, 25-27 August 2021

Virtual FENS Regional Meeting 2021
25-27 August 2021

Book of Abstracts

PTBUN Polish Neuroscience Society

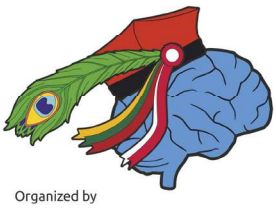
Lithuanian Neuroscience Association

IBRO PERC

FENS Federation of European Neuroscience Societies

Kraków

Honorary Patronage
of the Mayor of the City of Kraków
Jacek Majchrowski



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Organized by
the Polish Neuroscience Society and the Lithuanian Neuroscience Association

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WELCOME TO THE FENS REGIONAL MEETING 2021!

The FRM 2021 is organized jointly by the Polish Neuroscience Society (PNS) and the Lithuanian Neuroscience Association (LNA) under the auspices and with support from the Federation of the European Neuroscience Societies (FENS), and also with support from the International Brain Research Organization Pan-Europe Regional Committee (IBRO PERC). The FRM 2021 original city venue was Krakow, however, due to the COVID-19 pandemic, the Organizing Committee decided to hold the event on-line to ensure all attendees may meet safely.

The conference will present the latest developments in neuroscience research and host panel discussions on topics ranging from directions for future development to diversity issues in the academia. Traditionally, the Regional Meetings foster interactions among the researchers in the region. This year, we want to take the on-line format as an opportunity, and showcase neuroscience research in our region to the global community

Be a part of the FENS Regional Meeting 2021!

On behalf of the FRM Organizing and Scientific Committees,

Grzegorz Hess,
President of the Polish Neuroscience Society

Osvaldas Rukšėnas,
President of the Lithuanian Neuroscience Association

or following long-term food restriction (FR). The most prominent change observed was the age-related decrease in ApoE mRNA regardless of the food regimen applied. In animals kept on FR, this decrease was accompanied by an increase in the expression of HMG-CoAR and CYP46A1. In brief, food restriction reversed most of the dexamethasone-induced changes in the expression of genes involved in regulation of cholesterol homeostasis in aging rats, in a region-specific manner.

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A disclosure of conflicts of interest:
The authors declare that there is no conflict of interest

Agmatine protects mitochondria in LPS-stimulated microglia

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Mitochondria play a key role in energy metabolism and regulate some of the principal cellular processes such as the production of ATP and reactive oxygen species, as well as a regulation of apoptotic cell death. Mitochondrial dysfunction and oxidative stress are common threads in most neurodegenerative disorders, which are also accompanied by chronic microglial activation. Agmatine, neuromodulatory polyamine, was shown to exhibit neuroprotective effects in oxidative stress conditions. Therefore, the goal of this study was to determine the ability of agmatine to preserve mitochondrial function and prevent apoptosis during neuroinflammation.

The effects of 100 μM agmatine on cellular energy status and cell death were examined in LPS-stimulated BV2 microglial cell line. To detect changes in mitochondrial membrane potential, TMRE fluorescent assay was performed, while the changes in intracellular ATP concentration were determined by bioluminescent assay, 6h, and 24h after LPS stimulation. The expression of apoptosis regulators Bax and Bcl2 was assessed by Western blot analysis and the Bax/Bcl2 ratio was determined.

Agmatine increases mitochondrial membrane potential, indicating its protective role during mitochondrial insult caused by LPS stimulation. LPS and agmatine administrated separately, increase intracellular ATP levels, however, agmatine treatment followed by LPS stimulation enhances ATP production even further, at both time points. Moreover, agmatine shows an antiapoptotic effect by reduction of Bax/Bcl2 ratio in comparison to LPS stimulation.

We conclude that the results of this study indicate the capacity of agmatine to protect mitochondrial function and suppress apoptosis, which may be beneficial in neurodegenerative disorders and neuroinflammation.

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Conflict of interest: The authors declare that there is no conflict of interest.

Altered cerebellar output disrupts normal cortical oscillatory activity during movement and quiet wake

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The neural impulses responsible for accurate movement sequences are generated within the cerebello-thalamo-cortical motor circuit [1], that functionally group the structures involved in the motor act and present distinctive features depending on the motor behaviour. Here we aimed to identify the effect of altered cerebellar output on motor oscillations in the cortex during different motor states.

ECoG-EMG were bilaterally recorded on motor cortices and neck muscles, respectively, in Swiss albino mice. After a 90-min basal recording during a first day, recordings were done for 5 consecutive days, before (pre-kainate, 30-min) and after (post-kainate, 60-min) a daily kainic acid microinjection into the left cerebellar hemisphere, used to alter the cerebellar activity.

The epochs were classified by movement behaviour and dystonic phenotype. The results were computed into power spectral density (PSD) and coherence for each recording site.

The cortical PSD revealed a decrease in theta band after the cerebellar disturbance in both dynamic and quiet states, while beta power only dropped in the dystonic movements, suggesting that beta is sensitive to motor activity. The motor cortices coherence decreased in delta-theta and beta regardless of the motor behaviour. However, ECoG-EMG coherence revealed a decremental trend in beta and theta that was dependent to static behaviour, while motor activity triggered increased coherence in beta and low-gamma bands.

Our work showed that there are distinctive patterns of dystonic cortical oscillatory activity, which are dependent to motor behaviour, and others that are not sensitive to motor activity, suggesting a possible advance in describing dystonic ECoG patterns.