

FENS

REGIONAL MEETING

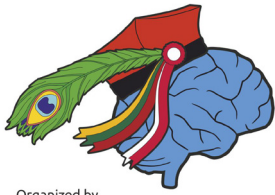
Kraków, Poland, 25-27 August 2021

Virtual FENS Regional Meeting 2021
25-27 August 2021

Book of Abstracts



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

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to be an effective target in the treatment of anxiety-related psychiatric disorders, but more clinical studies are still needed.

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Acknowledgments

The abstract was written without financial funding.

Declarations of interest

The authors have no conflict of interest to disclose.

NTPDase1/CD39 and Ecto-5'-nucleotidase/CD73 are Upregulated in a Sex-specific fashion in the Rat Fetal Brain After Repeated Antenatal Dexamethasone Treatment

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To accelerate organ maturation and prevent complications due to preterm birth, antenatal treatment with synthetic glucocorticoids (GCs – dexamethasone or betamethasone) is usually given between the 24th and 34th week of pregnancy to women at risk of delivery within the next seven days [1]. Despite recommendations, repeat courses of antenatal GCs are frequently given, although excessive GC stimulation may exert adverse neurodevelopmental effects [1]. The purinergic system is essential for neurodevelopment [2]. Extracellular purine levels are regulated by ectonucleotidases, with ectonucleoside triphosphate diphosphohydrolase 1 (NTPDase1/CD39) and ecto-5'-nucleotidase (e5'NT/CD73), abundant in the CNS, which jointly hydrolyze ATP to adenosine. Both ectonucleotidases are also involved in cell adhesion and migration [3]. We aimed to explore the effects of antenatal dexamethasone (DEX) treatment on the expression and enzymatic activity of NTPDase1/e5'NT tandem in the rat fetal brain. Wistar rat dams were treated with 0.5 mg/kg DEX, at gestation day (GD) 16, 17, and 18. We found sex-specific male-biased upregulation of CD39 and CD73 mRNA and protein abundances, and an increase in the corresponding

enzymatic activities in the rat fetal brain at GD21, induced by antenatal DEX treatment. Observed changes indicate a possible decrease in P2, and an increase in P1 purinergic receptors-mediated signaling, as well as a potential decrease in migration of progenitor cells, particularly pronounced in the brain of male fetuses. Together, sex-dependent induction of CD39 and CD73 might interfere with neurodevelopmental processes, thus contributing to adverse effects of antenatal DEX treatment, especially in males.

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Funding: This work was supported by the Ministry of Education, Science and Technological Development, the Republic of Serbia, contract No. 451-03-68/2020-14/200007 and 451-03-68/2020-14/ 200178 and the University of Defense (grant number MFVMA/04/19-21). J.S. received support from the Natural Sciences and Engineering Research Council of Canada (NSERC; RGPIN-2016-05867).

A disclosure of conflicts of interest: The authors declare no conflict of interest.

Neuroigin-2 regulates long-term GABAergic plasticity

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For decades, the research into synaptic plasticity was mainly focused on the excitatory synapses while GABAergic inhibitory transmission was thought to be devoid of complex long-term plasticity. This view changed recently when numerous plastic phenomena were discovered in GABAergic synapses (e.g. different forms of iLTP and iLTD). Nevertheless, our knowledge of adhesion proteins in GABAergic plasticity is still limited. Neuroigin-2 (NLG-2), locates specifically at the postsynaptic density, where it interacts with scaffold protein gephyrin and GABA_AR. Additionally, NLG-2 provides trans-synaptic adhesion through the binding of presynaptic neuexins. This study aimed to address the function of NLG-2 in GABAergic plasticity.

We recorded mIPSCs in CA1 pyramidal neurons in slices and induced iLTP using a short-term application of NMDA. Additionally, we used neuroilide-2, a peptide that blocks the interaction between NLG-2 and neuexins.

Obtained results show that when interactions between NLG-2 and neuexins are disrupted, NMDA-iLTP is abolished (control with scrambled peptide: 116% of baseline; neuroilide-2: 93%, n=5-6, p<0.001, 22 min after induction). Next, we asked about the time window of NLG-2 involvement in plasticity phenomenon.