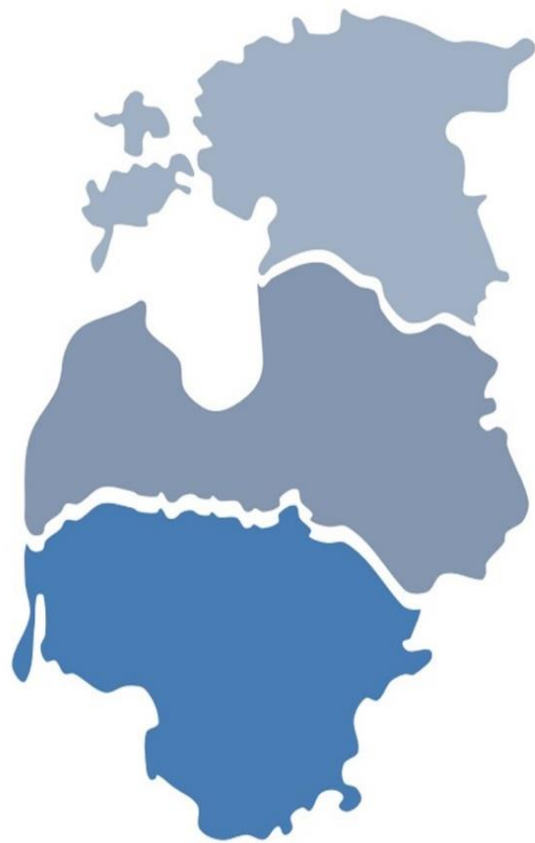


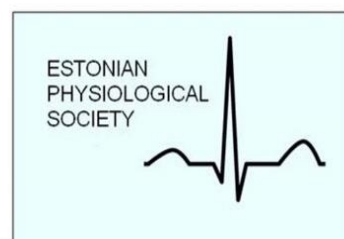
FEPS 2015

26 - 29 August
Kaunas, Lithuania



ABSTRACT BOOK

ACTA PHYSIOLOGICA
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16:30 S11-O2

Protective effect of alpha lipoic acid, aerobic or resistance exercise from colitis in passive cigarette smoking young rats

Yunus Basar¹, Ayse Cansu Berberoglu¹, Oktay Erkan¹, Anil Ozen¹, Tunahan Sen¹,
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16:45 – 17:15 COFFEE BREAK

14:15 – 16:45 Lecture hall 204

SYMPOSIUM 12:

Chloride homeostasis and neuronal function

Organiser: Michael Druzin

INVITED PRESENTATIONS

14:15 S12-1

Setting neuronal chloride gradient: a new role for extracellular matrix

Michael Druzin

Section for Physiology, Department of Integrative Medical Biology, Umeå University, Sweden.

14:45 S12-2

Impermeant anions, fixed charges, and the driving force of GABA_A R-mediated Cl⁻ currents

Juha Voipio

Department of Biosciences, University of Helsinki, Finland

15:15 S12-3

Presynaptic action potentials and GABA_A receptors

Dimitri M Kullmann

UCL Institute of Neurology, University College London, United Kingdom

**The Joint Meeting of the Federation of European Physiological
Societies and the Baltic Physiological Societies in Kaunas,
Lithuania, August 26-29, 2015**

ABSTRACTS

Protective effect of alpha lipoic acid, aerobic or resistance exercise from colitis in passive cigarette smoking young rats

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Aim: The role of passive cigarette smoking (PCS) and resistance exercise are not known. Our aim was to examine the effects of antioxidant ALA, chronic aerobic (AE) or resistance exercise (RE) on PCS rats with colitis.

Methods: Sprague-Dawley male rats (150-200g, n=54) were exposed to PSS (6 days/week, 4 cigarettes/day) while several groups were assigned to RE (climbing with weight; 3 days/week), others to AE (swimming; 3 days/week) whereas several groups were not exercised. Following 5 weeks, colitis was induced by intrarectal acetic acid. Subcutaneous ALA (50 mg/kg/day) or corn-oil were injected 3 days. Following decapitation, colon tissues were sampled to examine malondialdehyde (MDA) levels, myeloperoxidase (MPO), glutathione (GSH), superoxide dismutase, catalase activities, luminol and lucigenin chemiluminescence, macroscopic scoring, histologic examination. ANOVA and student's t test were used for statistical analysis.

Results: The increased macroscopic and microscopic scores, MPO, MDA, luminol, lucigenin measurements in colitis and PSS-colitis groups were decreased via ALA. ($p<0.05-0.001$). AE declined macroscopic and microscopic scores, MDA, lucigenin compared to colitis and PSS-colitis groups ($p<0.01-0.001$). AE with ALA decreased luminol ($p<0.05$). RE reduced microscopic score, MPO, MDA, luminol, lucigenin ($p<0.05-0.001$) that were increased with colitis. GSH levels were decreased ($p<0.01$) in PSS-colitis group while approaching control levels by ALA.

Conclusions: PSS and colitis induction increased inflammatory parameters and oxidant damage. Our results suggest that ALA, AE or RE might be protective in ulcerative colitis.

Symposium 12: Chloride homeostasis and neuronal function

S12-1

Setting neuronal chloride gradient: a new role for extracellular matrix

Michael Druzin

Section for Physiology, Department of Integrative Medical Biology, Umeå University, Sweden

Neuronal signaling relies on ion fluxes through membrane-bound channels. Such fluxes are allowed due to the transmembrane ionic gradients created and maintained by membrane transporters. The neuronal gradient for chloride, an anion mediating most of the inhibition in the mature CNS, is the result of activity of two chloride transporters working in opposite directions, NKCC1 moving chloride in and KCC2 moving chloride out of the cell. The KCC2 function may be both up- and down-regulated through a number of mechanisms which allow for the fine-tune adaptation to a varying transporting load which mostly depends on chloride influx through both synaptic and extrasynaptic inhibitory chloride-permeable ion channels such as GABAA- and glycine-receptors.

Besides intraneuronal factors affecting KCC2 function and, thus, the chloride gradient, there is a number of extra-neuronal factors suggested to have a great influence over the distribution of chloride across the neuronal membrane. The very recent experimental findings point to the extracellular matrix as an important player in regulation of intracellular chloride. It has been claimed that large anion groups located within the extracellular matrix set the chloride gradient due to the Gibbs-Donnan effect, thus, effectively sidelining KCC2 as a major contributor to the neuronal chloride homeostasis. However, our own experimental findings concerning chloride homeostasis in anterior hypothalamic neurons suggest quite a different mechanism of involvement of the extracellular matrix.

S12-2

Impermeant anions, fixed charges, and the driving force of GABA_AR-mediated Cl⁻ currents

Juha Voipio

Department of Biosciences, University of Helsinki, Finland

It has been recently suggested^{1,2} that electrostatic (Donnan) effects of immobile anions, not the cation-chloride cotransporters³ (CCCs) KCC2 and