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Aim: The aim of this article is Hydroalcoholic extract of *Dorema aucheri* leaves prevents weakening of the brain antioxidant defense system and inhibits oxidative damage in rat model of ischemic stroke

Introduction: The production of free radicals is the principal mechanism of brain injury in ischemic stroke. The present study tried to identify whether pretreatment with hydroalcoholic extract of *Dorema aucheri* (DA) leaves potentiates the brain antioxidant system and decreases brain infarction and oxidative damage during cerebral ischemia–reperfusion.

Methods: Three groups of rats were randomly selected (each group; $n = 12$); sham, control ischemic and ischemic pretreatment groups. Treated rats received freshly hydroalcoholic extract of DA (200 mg/kg/day) for 14 days. Then, cerebral ischemia–reperfusion was achieved by 90 minutes middle cerebral artery (MCA) occlusion followed by 24 h reperfusion. Infarct volume and contents of malondialdehyde (MDA), glutathione and nitrate (NOx) as well as superoxide dismutase (SOD) and catalase activities were assessed after 24 h reperfusion

Results: The contents of MDA and nitrate significantly increased in the ischemic hemispheres by 34% and 14%, respectively. Brain ischemia decreased the glutathione content (20%) and activities of catalase (38%) and SOD (14%) in ischemic hemispheres compared to sham rats. Treatment with DA before MCA occlusion significantly decreased the infarction in cortex and striatum by 63% and 75%, respectively, compared to control. DA considerably reduced the contents of MDA and nitrate in ischemic hemispheres by 28% and 11%, respectively, compared to control rats. Treatment with DA also increased the glutathione content (7%) and activities of catalase (46%) and SOD (16%) of ischemic hemispheres.

Conclusion: The present study revealed that pretreatment with hydroalcoholic extract of DA leaves prevents weakening of the brain antioxidant defense system and decreases the brain damage during cerebral ischemia–reperfusion.

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PS150

The Levator Auris Longus (LAL) muscle as an accessible system to study the effects of Botulinum Toxins in vivo

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Aim: In the present work, we aimed to find a reproducible model to study the effects of Botulinum neurotoxins (BoNTs), that allowed a widespread visualization of the intoxicated nerve terminals.

Introduction: Despite the well-established successful use of BoNTs to treat a variety of human conditions, their mechanism of action is still not fully understood. Thus, there is an emergent need of new and accurate models to study the effects of BoNTs. However, considering their potential lethality, it is challenging to find reproducible models to study the local application of BoNTs in living animals that allow a widespread visualization of the intoxicated nerve terminals.

In these work, we studied the innervation pattern and the effect of BoNTs in a group of small subcutaneous cranial muscles that are responsible for moving the pinna in rodents. Although all are easily accessible and manipulated, we focused on the levator auris longus (LAL).

Methods: Animals were injected subcutaneously with the indicated doses of BoNT/A, in the cranial muscles area. Muscles were then dissected and prepared for wholemount staining for Synapsin-I, cleaved SNAP-25 (synaptosome-associated protein of 25 kDa) and β 3-tubulin.

Results: Detection of cleaved SNAP-25, the end-product of the catalytic action of BoNT/A, was possible even with injections as low as 0.1 ng. Mapping of the injected muscle showed the effect of BoNT/A in the majority of the endplate population. Also, seven days after BoNT/A injection, a sprouting process was evident, a landmark of regeneration.

Conclusion: BoNTs delivery to the LAL is a sensitive, simple and reproducible model to study the mechanisms of action of these toxins as it allowed the evaluation of BoNT/A effects throughout the entire muscle, without sampling bias. Thus, we forward that the LAL manipulation may constitute an excellent model to clarify the mechanisms of action of BoNTs in the neuromuscular system.

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PS188

NLRP3 inflammasome as a potential target to reduce epileptic-like activity



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Aim: Decipher how inflammation drives epilepsy and how NLRP3 targeting impacts epileptic-like activity.

Introduction: Epilepsy is one of the most common neurological diseases in worldwide. Inflammation was linked to the presence of inflammasomes, cytosolic multiprotein complexes, which promote the release of proinflammatory cytokines, namely IL-1 β . Although a feedback loop has been described between inflammation and epilepsy, the role of inflammasomes in epilepsy is still unknown. NLRP3 is the most studied inflammasome,¹ activated by a two-signal process: 1) a priming signal (as lipopolysaccharides – LPS), which enhances the expression of NLRP3 and pro-IL-1 β ; and 2)

an activating signal (as ATP), which promotes the formation of the complex.

Methods: Organotypic slices were used to assess the interplay between inflammation and epilepsy. Slices were exposed to different concentrations of LPS (5, 10 and 20 ng/mL), either alone or in the presence of ATP (1 mM). LPS-induced inflammation was characterized using molecular-based assays, such as ELISA to quantify IL-1 β , CBA to measure TNF- α , and western blot to assess the expression of Iba-1, GFAP, NLRP3/ASC, and α II-Spectrin. Field potential recordings were used to evaluate the epileptic-like activity of the slices and the effect of MCC950, a NLRP3 selective inhibitor,² was assessed.

Results: Results obtained by ELISA showed a significant increase in IL-1 β concentration in slices exposed to 10 ng/ml LPS/1 mM ATP. TNF- α , assessed by CBA, was also significantly increased in this condition, corroborating the inflammatory phenotype. No changes in NLRP3 expression were observed by immunoblot analysis, but ASC, one component of the inflammasome, showed a decreased expression in LPS/ATP exposed slices, suggestive of its binding to NLRP3 and thus to complex formation.

Furthermore, epileptic-like activity, measured by field potential recordings, was blocked by MCC950 (10 μ M).

Conclusion: We demonstrate that LPS induces an inflammatory phenotype in organotypic slices. NLRP3 blockade eliminated the epileptic-like activity of the slices.

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PS110

Study of the modulatory CNS regions in the visual circuit Retina-Superior Colliculus-Lateral Posterior nucleus triggering freezing behavior



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Aim: The goal is to understand the neuronal networks organization from the sensory input to the freezing behavior through the identification of modulatory brain regions that project to the Superior Colliculus.

Introduction: The behavior of an animal can be triggered by signals in its visual environment. Threatening visual stimulus evoked innate defense behaviors as freezing behavior. This project is focused in one visual-guided behavioral circuit that links the retina visual information with the Lateral Posterior thalamic nucleus(LP) via Superior Colliculus(SC).

Methods: The experimental approach is based on retrograde viral tracing techniques. Using the stereotaxic surgery, the first injection with a Herpes Simplex Virus expressing TVA receptor and glycoprotein G was done at LP. After 21 days, the second injection was done at the SC with a Rabies Virus coated by EnvA and lacking of glycoprotein G. The combination of these viruses allowed the restriction of the viral tracing to the circuit of interest. Subsequently, the experimental procedure continued perfusing the mouse, slicing the brain and staining it. Finally, the slices were scanned using the fluorescent confocal microscope.

Results: The resulting images presented labeled cells in all brain areas that sent inputs to collicular neurons that are projecting to LP. The main nuclei identified were the Periaqueductal gray, the primary visual cortex and the Substantia nigra, suggesting their modulatory role in freezing responses.

Conclusion: The main areas labeled are sending excitatory projections to SC to reinforce the freezing behavior. Also, Ntsrl-GN209-Cre mice used in combination with flox-HSV for the first injection restricted more the viral tracing, specifically to the Ntsrl+ Wild-field neurons of SC which were already known that project to LP. The results were not completely consistent with the non-flox-HSV injections but the main nuclei named above were also labeled. These results suggest that the flox-HSV is necessary to exclude nonspecific labeling of projections from SC-LGN.

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PS048

The influence of antipsychotics therapy and sociodemographic characteristics on cognitive performances in acute phase of schizophrenia



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Aim: The main purpose of this research was to examine the influence of sociodemographic characteristics (gender, age, level of education, heredity, alcohol and psychoactive substances), and the effect of different therapies on cognitive capabilities of patients diagnosed with schizophrenia.

Introduction: Schizophrenia, as one of the most common psychiatric diseases, is characterized by generalized cognitive damage with various degrees and in all domains of cognitive functioning. Cognitive dysfunction is one of the main causes of poor social and professional functioning for patients with schizophrenia.

Methods: The research involved 50 patients with acute phases of schizophrenia from the Psychiatric Clinic in Novi Sad. The primary instrument for the research was the standardized test for examination of cognitive impairments, Mini-Mental Scale Examination (MMSE).

Results: Acquired data correlated with MMSE score, noting the degree of cognitive impairments in patients, particularly significant with relation to age and duration of illness. Gender, level of education and type of used antipsychotics were not significantly correlated with MMSE score.

Conclusion: During this research it is found that aging and longer illness duration bear significant correlation to higher levels of cognitive impairment.

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PS190

Voluntary inhibition of saccadic eye movements: EEG study



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