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.

http://dx.doi.org/10.1016/j.pbj.2017.07.073

PS150

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

Torrado Marília1,2,3, Avelino António1,2, Cruz Célia Duarte1,2, Leonor Rodrigues2,3

1 Departamento de Biomedicina – Unidade de Biologia Experimental, Faculdade de Medicina da Universidade do Porto, Portugal
2 Translational NeuroUrology, IBMC – Instituto de Biologia Molecular e Celular, Universidade do Porto, Portugal
3 Instituto de Investigação e Inovação em Saúde – i3S, Universidade do Porto, Portugal

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-1ß. 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, activated by a two-signal process: 1) a priming signal (as lipopolysaccharides – LPS), which enhances the expression of NLRP3 and pro-IL-1ß; and 2) a response signal, which is independent of the priming signal and involves cleavage of gasdermin D (GSDMD), a cellular apoptosis effector.