Introduction: The model of accelerated senescence with the administration of d-galactose is used in anti-aging studies. However, reports have questioned its effectiveness. To clarify this issue, we used high-dose d-galactose on young rats and studied the immature granule cells stained with the neurogenesis marker doublecortin (DCX). We also used EGCG, a green tea catechin, to verify if there are neuroprotective effects in the d-galactose-treated animals.

Methods: At 4 weeks of age, male Wistar rats were allocated to a control group (n = 7), a d-galactose group (300 mg/kg body weight, intraperitoneally) (n = 5; GAL) and to a d-galactose + EGCG (oral solution, 2 grams/L) group (n = 5; gal + EGCG) during 4 weeks. After this period DCX immunocytochemistry was performed. The dendritic trees of immature granule cells were drawn with the aid of a camera lucida and a metric analysis of the dendritic segments of the dendritic trees was performed.

Results: No differences in all parameters quantified were found when controls and gal rats were compared. However, the results show that the total dendritic length of the dendritic trees of gal + EGCG rats was significantly reduced when compared with controls (p < 0.03). There were no differences in the others dendritic parameters quantified.

Conclusion: d-Galactose did not induce disturbance of the neurogenesis as shown by the absence of alterations in the dendritic trees confirming our previous studies. Surprisingly, the addition of EGCG led to a reduced total dendritic length. This unexpected effect can be explained if we consider that the addition of the catechin acted as a second aggression leading to a disturbed dendritic tree of the immature neurons.

Acknowledgements: This article was supported by ERDF through the operation POCI-01-0145-FEDER-007746 funded by the Programa Operacional Competitividade e Internacionalização – COMPETE2020 and by National Funds through FCT - Fundação para a Ciência e a Tecnologia within CINTESIS, R&D Unit (reference UID/IC/4255/2013).

Aim: Evaluate thrombophilia causing genetic variants and ACE gene I/D variant impact on patients with ischemic stroke.

Introduction: Every year, 15 million people worldwide suffer a stroke that is the second leading cause of disability. Genetic variants in Leiden factor coding gene (F5) and prothrombin gene (F2) cause inherited thrombophilia which is associated with increased risk of intravascular thrombosis, thromboembolism and cerebral stroke. Angiotensin-converting enzyme (ACE) coding gene I/D variant is discussed among numerous conditions including stroke.

Methods: In the study there were included 115 patients with mean age 70.3 ± 11.0 years, with diagnosed ischemic stroke. Control group for F5 and F2 gene variations consisted of 124 individuals with mean age 55.6 ± 14.6 years. And for ACE gene variation 248 individuals with mean age 56.8 ± 11.4 years. DNA was extracted from peripheral blood using standard phenol-chloroform method. Genotyping of F5 gene variant G1691A and F2 gene variant G20210A was performed using PCR-RFLP. ACE gene I/D variant genotyping were performed using PCR. Statistical analysis was performed using Fisher’s exact test and SPSS v22.0 software.

Results: F2 gene variant were more frequent in patient group. Frequency in patients were 0.017 and in control group 0 (p = 0.038). F5 gene variant frequency in both patients and control group were 0.012 (p > 0.05). Seven patients (5.6%) had one variant in one of coagulation factors encoding genes comparing to three in control group (2.4%) (p > 0.05). Mean age for patients with identified variations in F2 or F5 was not significantly different comparing to other patients (p > 0.05). ACE gene I/D genotypes and allele frequencies in stroke patients were not significantly different from controls – I allele frequencies were 0.452 in patients versus 0.470 in controls (p > 0.05).

Conclusion: Prothrombin encoding gene variant G20210A could be risk factor for ischemic stroke. F5 and ACE gene I/D genotypes are not associated with ischemic stroke.
Effect of resveratrol on the cartilage and nociceptive system of Osteoarthritic animals


Department of Biomedicine - Experimental Biology Unit, Faculty of Medicine of the University of Porto, Porto, Portugal

Aim: This study aims to evaluate the effect of RV on the nociceptive behavior, histopathological alterations at the knee and DRG neurons of OA rats.

Introduction: Osteoarthrosis (OA) is a common degenerative joint disease and arthritic pain is a prominent symptom associated with reduced quality of life. Peripheral pain mechanisms seem to be involved, with cartilage lesions showing a repercussion in Dorsal Root Ganglia (DRG) neurons. Resveratrol, a polyphenol with proven anti-inflammatory, anti-oxidant and neuroprotective properties, has been shown to prevent development of OA and act as an antinociceptive agent. However, its systemic effects when tested in vivo, and its potential to be used in mitochondria-targeted formulations to modulate the pathophysiological changes underlying AD from their early stages.

Methods: The polyphenolic profile of elderberry extract and of anthocyanin-enriched fraction was evaluated by HPLC-DAD, the optical properties by UV–vis and fluorescence spectroscopy and the reduct behavior by cyclic voltammetry. Antioxidant properties were assessed in cell-free assays while the ability the elderberry extract to modulate the mitochondrial reoxid chain was evaluated in rat brain mitochondria.

Results: HPLC analyses showed that elderberry extract is a mixture of chemical compounds, particularly rich in anthocyanins. It exhibits intrinsic fluorescence properties with potential for bioimaging. Reversible reduct behavior and ability to scavenge DPPH, nitric oxide and superoxide radicals. The antioxidant, optical and redox properties of elderberry extract are strongly correlated to their content in anthocyanins. Bioenergetic studies show that elderberry extract has ability to promote the oxidation of NADH in aqueous phase and deliver electrons to ubiquinone or complex III in the inner-mitochondrial membrane, overcoming the complex I inhibition promoted by rotenone.

Conclusion: Elderberry anthocyanins have potential to be used in mitochondria-targeted formulations to modulate the patho-physiological changes underlying AD from their early stages.

Acknowledgements: This work received financial support from National Funds (FCT/MEC) through project UID/QUI/50006/2013, co-financed by FEDER through COMPETE, under the Partnership Agreement PT2020, and from NORTE 2020, under the PORTUGAL 2020 Partnership Agreement, through ERDF (NORTE-01-0145-FEDER-000024).

http://dx.doi.org/10.1016/j.pbj.2017.07.066

PS016

Bupivacaine treatment enhances the regeneration of the lesioned external urethral sphincter of the rat

J.P. Morais, M. Torrado, A. Avelino

1 Escola das Ciências da Vida e Ambiente, Universidade de Trás-os-Montes e Alto Douro
2 Department of Biomedicine, Experimental Biology Unit, Faculty of Medicine, University of Porto, Portugal
3 Translational NeuroUrology Group, IBMC – Instituto de Biologia Molecular e Celular, Porto, Portugal
4 i3S - Instituto de Investigação e Inovação em Saúde, Porto, Portugal

E-mail address: joaopfmmorais@gmail.com (J.P. Morais).

Aim: In this study we intend to verify if bupivacaine treatment can be used to enhance the repair of the lesioned urethral sphincter in rat.

Introduction: Stress urinary incontinence (SUI) is a major and frequent urinary dysfunction. It has been associated with external urethral sphincter (EUS) weakness due to several causes. Among them, ischemia and nerve lesion frequently associated with childbirth. The current treatments are mainly surgical but are far from being satisfactory. The local anesthetic bupivacaine is known to exert myotoxic action, followed by muscle regeneration with increased strength. This effect was already used in ocular muscles to treat strabismus. In the present study we evaluated the effect of bupivacaine application in the recovery of the damaged EUS.

Methods: A lesion of the external urethral sphincter (urethrolysis) was performed in adult female Wistar rats using established protocols. Two weeks after the lesion, the animals were injected in the EUS with 0.4 ml of 0.5% bupivacaine. Ten days later, the whole urethra was removed, fixed and sectioned in paraffin wax. Sections