

¹ Department of Biomedicine – Unit of Anatomy, Faculty of Medicine, University of Porto, Alameda Prof. Hernâni Monteiro, 4200-319 Porto, Portugal

² Center of Health Technology and Services Research (CINTESIS), Faculty of Medicine, University of Porto, Rua Dr. Plácido da Costa, 4200-450 Porto, Portugal
E-mail address: fbarreto44@hotmail.com (F. Barreto).

Aim: In the present study, we aimed to explore the effect of D-galactose administration and epigallocatechin-3-gallate (EGCG) on the dendritic trees of developing granule cells of the hippocampal formation (HF) of young male rats.

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).

<http://dx.doi.org/10.1016/j.pbj.2017.07.064>

PS079

Analysis of variations in the F5, F2 and ACE genes among Latvian patients with ischemic stroke

Anna Inese Tutāne

Rīga Stradiņš University, Latvia

E-mail address: annatutane@gmail.com.

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 in 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.

<http://dx.doi.org/10.1016/j.pbj.2017.07.065>

PS205

The bioactive compounds from elderberry to modulate mitochondrial dysfunctions underlying Alzheimer's disease



Dina Neves^{1,*}, João Bernardo¹,
Patrícia Valentão¹, Maria C. Oliveira²,
David M. Pereira¹, Paula B. Andrade¹,
Romeu A. Videira¹

¹ REQUIMTE/LAQV, Laboratório de Farmacognosia, Departamento de Química, Faculdade de Farmácia, Universidade do Porto, Rua de Jorge Viterbo Ferreira, N.º 228, 4050-213 Porto, Portugal

² Centro de Química de Vila Real (CQ-VR), Departamento de Química; Escola de Ciências da Vida e do Ambiente, Universidade de Trás-os-Montes e Alto Douro (UTAD), P.O. Box 1013, 5001-801 Vila Real, Portugal

E-mail address: up201302607@ff.up.pt (D. Neves).

Aim: The specific objective of this work is to establish a correlation between the physical-chemical properties of the aqueous extract of elderberry (*Sambucus nigra* L.) and its ability to tune the cell redox state and to overcome mitochondrial dysfunctions, which are pathological events with high relevance in Alzheimer's disease (AD).

Introduction: Currently, there is no effective medicine to prevent or delay the progressive brain degeneration underlying cognitive decline and dementia that characterize AD. Previous works support the idea that the loss of mitochondrial functionality, connected with the decline of complex I activity, is able to promote AD phenotype through the activation of multiple pathophysiological pathways, including oxidative stress, neuroinflammation, and also tau and amyloid-beta pathologies. Thus, multi-targeted

