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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.

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PS079

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

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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.

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PS205

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



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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



approaches supported by mixtures of natural bioactive compounds should reveal more effectiveness than classical therapeutics for AD.

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 redox behavior by cyclic voltammetry. Antioxidant properties were assessed in cell-free assays while the ability the elderberry extract to modulate the mitochondrial redox 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 redox 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 pathophysiological changes underlying AD from their early stages.

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PS016

Effect of resveratrol on the cartilage and nociceptive system of Osteoarthritic animals



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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: Osteoarthritis (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 once the disease has fully developed remain unclear.

Methods: To evaluate this, OA was induced in 18 male Wistar rats through intra-articular injection of mono-iodoacetate (MIA) (day 0). Animals were allowed to develop the disease for two weeks,

after which followed a 4-week-long treatment with resveratrol or vehicle, administered intraperitoneally twice daily (10 mg/kg). Nociceptive behavior was quantified weekly using the CatWalk and Knee-Bend tests. Animals were sacrificed one week after the last treatment administration, their knees were dissected for histopathological analysis, and the DRG were dissected and processed for immunohistochemical evaluation of activating transcription factor 3 (ATF-3) neuronal expression.

Results: Resveratrol was unable to prevent cartilage degeneration but it significantly decreased ATF-3 expression. The nociceptive behavior of OA animals treated with resveratrol decreased during the first three weeks of treatment, in comparison to day 14 (before treatment was initiated), as shown by Knee-Bend scores. However, this tendency reverted as the disease progressed.

Conclusion: These results indicate that resveratrol may have antinociceptive effects in the early stages of the disease development, but it might not play such a relevant role once the disease has progressed. Thus, further studies are needed to fully understand the possible role of resveratrol in the different stages of OA.

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PS173

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



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Aim: In this study we intent 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 child-birth. 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 (urethrol-ysis) 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