blecortin (DCX). We also used EGCG, a green tea catechin, to verify
ever, reports have questioned its effectiveness. To clarify this issue
d on the dendritic trees of developing granule cells of the hippocam-
d formation (HF) of young male rats.

**Methods:** At 4 weeks of age, male Wistar rats were allocated
to a control group \((n = 7)\), a \(\beta\)-galactose group \((300 \text{ mg/kg body weight, intraperitoneally}) (n = 5; \text{ GAL})\) and to a \(\beta\)-galactose + EGCG (oral solution, 2 grams/L) group \((n = 5; \text{ gal + EGCG})\) 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:** \(\beta\)-Galactose did not induce disturbance of the neuronogenesis 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 acts as a second aggression leading to a disturbed dendritic tree of the immature neurons.

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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 antiinociceptive 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 intraperitoneal 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 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