if there are neuroprotective effects in the blecortin (DCX). We also used EGCG, a green tea catechin, to verify we used high-dose 

dgalactose administration and epigallocatechin-3-gallatte (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

**PS205**

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

Dina Neves 1, João Bernardo 1, Patricia Valentão 1, Maria C. Oliveira 2, David M. Pereira 1, Paula B. Andrade 1, Romeu A. Videira 1

1 REQUIMTE/LAQV. Laboratório de Farmacognosia, Departamento de Química, Faculdade de Farmácia, Universidade do Porto, Rua de Jorge Viterbo Ferreira, N° 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