blecortin (DCX). We also used EGCG, a green tea catechin, to verify

ever, reports have questioned its effectiveness. To clarify this issue

dadministration of

dendritic formation (HF) of young male rats.

on the dendritic trees of developing granule cells of the hippocam-

d. We also used EGCG, a green tea catechin, to verify
if there are neuroprotective effects in the d-galactose-treated ani-

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 con-
ts. There were no differences in the others dendritic parameters quantified.

Conclusion: d-Galactose did not induce disturbance of the neu-
rogenesis 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 cat-
echin 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 Internacionaliza-
ção - COMPETE2020 and by National Funds through FCT - Fundaç ç a
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

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Aim: The specific objective of this work is to establish a correlation
between the 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 mitocondrial functionality, connected with the decline of complex I activity, is able to promote AD phenotype through the activation of multiple pathophysio-

 logos pathway, including oxidative stress, neuroinflammation, and also tau and amyloid-beta pathologies. Thus, multi-targeted

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 vari-
ants 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 vari-
ant 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. Con-
trol 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 per-
fomed 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
cogulation factors encoding genes comparing to three in control
(p > 0.05). Mean age for patients with identified varia-
tions 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 geno-
types are not associated with ischemic stroke.

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