**Results:** Immobilisation time (IT) in FST after the administration of imipramine was shorter than the control, same as for subgroups treated with AE I, II and VS. In the subgroup treated with fluoxetine, IT in TST was shorter than the control time, and the same was observed in subgroups treated with AE I, II and VS.

Significant binding energies were found for Serotonin Reuptake Transporter (SERT) and venlafaxine (−7.20 kcal/mol) and verbacoside (−6.61 kcal/mol), and for the Leucine Transporter (LeuT), the homologue of the noradrenaline reuptake transporter, and verbanalin (−6.27 kcal/mol) and caffeine acid (−5.85 kcal/mol).

**Conclusion:** In both pharmacodynamic tests the antidepressive effect of AE and VS has been confirmed. Verbenalin and verbacoside binding energies and poses in interaction with SERT were similar to those of paroxetine. For LeuT, verbanalin showed both a similar binding energy and pose to that of imipramine, whereas caffeine acid showed only a similar binding energy.1–4

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**PS231**

**Effects of Vitamin D on the expression of markers of principal neurons, interneurons and astrocytes in cerebral cortex and hippocampus in gerbils exposed to transient global cerebral ischemia**

M. Malinic1, G. Jevtic Dozudic

Institute of Clinical and Medical Biochemistry, Faculty of Medicine, University of Belgrade

E-mail address: marija.malinic@gmail.com (M. Malinic).

**Aim:** Examination of the effects of vitamin D pretreatment on the expression of markers of principal neurons (NeuN), inhibitory interneurons (PV) and astrocytes (GFAP) in cerebral cortex and hippocampus in gerbils who were exposed to transient global cerebral ischemia.

**Introduction:** Brain ischemia may cause serious damage to the cells in the central nervous system. Vitamin D has an important role in brain injury treatment due to its neuroprotective effects.

**Methods:** Gerbils were divided in 5 groups: control group; two groups that underwent ischemia and then reperfusion for three (I/R3d) and seven days (I/R7d) and two groups that were treated with vitamin D before I/R (vitD + I/R3d and vitD + I/R7d). Complete blood supply to the brain was cut off for 10 minutes and reperfusion lasted 3 and 7 days. They were daily treated with vitamin D for 7 days prior ischemia. Expression of proteins was detected using Western blot.

**Results:** No changes were detected in expression of NeuN markers in cortex of experimental groups, while there was increase in hippocampus in groups I/R7d and vitD + I/R7d in comparison to the control group and group vitD + I/R3d. Expression of PV in cortex was significantly reduced in group I/R7d in comparison to group I/R3d, whereas in hippocampus the expression was significantly higher in group vitD + I/R3d than in group I/R3d. Expression of GFAP has significantly risen in all groups in comparison to the control group whereas in hippocampus there was a rise in groups vitD + I/R3d, I/R7d and vitD + I/R7d in comparison to the control group. There was also a rise of GFAP expression in groups treated with vitamin D (vitD + I/R3d and vitD + I/R7d) in comparison to those that have not been treated (I/R3d, I/R7d).

**Conclusion:** Vitamin D has positive effect on astrocytes in both structures of gerbils that underwent global cerebral ischemia, especially in hippocampal region.

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**PS238**

**Identification of genetic modifiers of somatic CAG instability in Huntington’s Disease by in vivo CRISPR – Cas9 genome editing**

A. Azevedo1,2, M. Kovalenko2, M. Andrew2, F. Zhang3, J. Lee2,4, V. Wheeler2,4, R. Mouro Pinto2,4,∗

1 University of Porto
2 Center for Genomic Medicine, Massachusetts General Hospital

**Abstract:** Huntington’s disease (HD) is a neurodegenerative disorder that is caused by an expansion of a trinucleotide repeat (CAG) in the huntingtin gene (Huntingtin). This expansion creates a toxic protein that leads to neuronal death and HD motor symptoms, which include chorea, dementia, and psychiatric symptoms. The pathology of HD is characterized by accumulation of a toxic polyglutamine (PolyQ) fragment in neurons, and the severity of the motor symptoms is directly related to the size of the CAG repeat. Current treatments for HD have limited efficacy and are aimed at managing symptoms. Genetic modifiers of CAG expansion may offer potential therapeutic targets for this neurodegenerative disease.

**Aim:** To identify genetic modifiers of somatic CAG instability in HD using the CRISPR-Cas9 genome editing system.

**Methods:** We developed a transgenic mouse model by delivering a Cas9-expression vector using an adenoviral vector. CRISPR-Cas9 genome editing was performed in brain tissues of transgenic mice to create a somatic CAG instability model. The CAG expansion was monitored using a fluorescent reporter. Genetic modifier screens were performed by crossing mice with a CAG-expanded vector to identify genes that modulate somatic CAG expansion.

**Results:** We identified several genetic modifiers of somatic CAG instability in HD. These modifiers include genes involved in DNA repair, RNA metabolism, and protein degradation pathways.

**Conclusion:** These genetic modifiers may represent potential therapeutic targets for HD.

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