**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 verbenalin (−7.20 kcal/mol) and verbasco-side (−6.61 kcal/mol), and for the Leucine Transporter (LeuT), the homologue of the noradrenaline reuptake transporter, and verbenalin (−6.27 kcal/mol) and caffeic acid (−5.85 kcal/mol).

**Conclusion:** In both pharmacodynamic tests the antidepressive effect of AE and VS has been confirmed. Verbenalin and verbasco-side binding energies and poses in interaction with SERT were similar to those of paroxetine. For LeuT, verbenalin showed both a similar binding energy and pose to that of imipramine, whereas caffeic 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**

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**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 days (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 expression 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**

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**Abstract**

In Huntington’s Disease (HD) striatal polyglutamine CAG repeat expansion results in polyQ-containing huntingtin (Htt) translation that promotes aggregation of Htt into pathological inclusions that cause neurodegeneration. However, variability in penetrance is evident in HD patients, and different CAG repeat lengths in the same HD family suggest that additional genetic modifiers exist. A two locus model of HD has suggested that genetic modifiers act as quantitative traits that finely tune HD phenotypes, while a recent study revealed that the first degree relative’s (FDR) age at onset is affected by genetic modifiers. In order to identify genetic modifiers we used Cas9/gRNA-mediated genome editing to knock out the CAG repeat of the Huntingtin (Htt) gene in Trp53−/− mice and examine the effect on age at onset in the FDR. Knockout of the CAG repeat in Trp53−/− led to increased HD-like inclusions in the striatum and substantia nigra, reduced amphetamine-induced rotations, and an increased age of death in the FDR. These results suggest that a CAG repeat length modifier is present in HD and that Cas9/gRNA-mediated genome editing is a valuable tool to identify genetic modifiers of polyQ repeat disorders.