**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 Transpeter (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 (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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**Aim:** CAG repeat instability is a critical feature of Huntington’s Disease (HD). HD is caused by a somatic expansion of the CAG repeat, which contributes significantly to the disease phenotype. The aim of this work is to identify genetic modifiers of somatic CAG repeat instability in HD in vivo.

**Introduction:** CAG repeat instability is a clinical hallmark of HD. In order to study the in vivo impact of genetic modifiers of somatic CAG repeat instability in HD, the CRISPR/Cas9 system was used to introduce somatic CAG repeat instability into the HD mouse model.

**Methods:** Male Wistar rats were injected with scopolamine for 14 consecutive days in order to induce drug model of dementia. A DSC study was performed to evaluate the effect of scopolamine on different brain segments using DSC.

**Results:** The DSC measurements revealed large differences between the denaturation profiles of rat brain supernatants and blood serum. The thermograms of brain tissues displayed clearly expressed low-temperature exothermic transitions with peaks in the range 35–45 °C which are missing in blood serum samples. There were differences between the thermograms of the separate brain segments as well. The thermodynamic parameters of the denaturation profiles were also determined.

**Conclusion:** These measurements show that DSC is an appropriate method with great potential for detection and characterization of the changes taking place at molecular level in different tissues, especially in brain tissues affected by neurodegenerative disorders.