**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

**References**

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**Aim:** Examination of the effects of vitamin D pretreatment on the expression of markers of principal neurons, interneurons and astrocytes (GFAP) in cerebral cortex and hippocampus in gerbils 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.

**Acknowledgements:** This research was made with the support of Science Fund at Medical University–Sofia, Bulgaria, Project 8-C/2016 and National Science Fund Project DN03/13/2016.

http://dx.doi.org/10.1016/j.pbj.2017.07.081

**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.

http://dx.doi.org/10.1016/j.pbj.2017.07.082

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

**Introduction:** Huntington’s Disease (HD) is a progressive neurodegenerative disorder caused by an expansion of the polyglutamine repeat in a protein coded by the huntingtin gene. It is characterized by movement disorders, cognitive dysfunction, and psychiatric symptoms. CAG expansion in the huntingtin gene results in an unstable polyglutamine tract in the huntingtin protein, which leads to aggregation of huntingtin inclusions that cause neuronal degeneration.

**Hypothesis:** Genetic modifiers that contribute to the expression of CAG instability can be identified through in vivo CRISPR genome editing.

**Materials and Methods:**

1. **Cell Culture:** Human embryonic stem cells (hESCs) were used as a model system for CAG instability studies. The huntingtin gene was modified using CRISPR-Cas9 technology.
2. **CAG Instability Assay:** The CAG expansion in the huntingtin gene was measured using fluorescence in situ hybridization (FISH) and qPCR.
3. **Gene Expression Analysis:** Microarray analysis was performed to identify gene expression changes associated with CAG instability.
4. **Statistical Analysis:** Statistical analysis was performed using R software to identify significant gene expression changes.

**Results:**

1. **CAG Instability Measurement:** The CAG expansion in the huntingtin gene was successfully measured using FISH and qPCR.
2. **Gene Expression Analysis:** Microarray analysis identified several gene expression changes associated with CAG instability.

**Conclusion:** Genetic modifiers that contribute to the expression of CAG instability were identified through in vivo CRISPR genome editing. These findings can provide insights into the molecular mechanisms underlying Huntington’s Disease.

http://dx.doi.org/10.1016/j.pbj.2017.07.082