Aim: The aim of our study was to find out EEG markers of inhibitory control in human.

Introduction: The voluntary inhibition is an important component of cognitive control. It is strong in healthy adults and weak in people with schizophrenia. The cortical mechanisms of inhibition are associated with event-related potentials (ERPs). In the case of a saccadic response some new EEG correlates of inhibition could be found.

Methods: Sixteen healthy right-handed subjects (18–22 years) participated in the study. We used a modified “Go/No go delay” paradigm with long interstimulus interval (2800–3000 ms). The task involved two types of target stimuli (“Go”, “No go”) with 50% probability. EEG and saccades were recorded simultaneously. ERPs were determined by means of coherent averaging relative to target stimulus onset. The EEG brain mapping was used to depict spatial dynamics of P1.

Results: P1 peak latency was 90–140 ms and tended to increase in cases of inhibition (by 6 ± 0.5 ms, p < 0.05). In the “No go” situation P1 amplitude was significantly lower than that in case of “Go” stimulus presentation (by 3.3 ± 0.7 mkV, p < 0.05). Regardless of the place where “No go” stimulus appeared, P1 amplitude was significantly higher on the right hemisphere, that is known to be the dominant one for inhibitory control. The EEG mapping data demonstrate the “bottom-up” spreading of P1 foci in “No go” conditions. It also indicates inhibitory processes.

Conclusion: The spatiotemporal parameters of P1 component in “Go/No go delay” paradigm reflect inhibitory processes. Therefore, P1 can be used as EEG marker of inhibitory violations in the clinical research. Our current research involves as subjects the patients with schizophrenia and ultra-high risk patients, as they demonstrate weakened the inhibitory processes. The data would contribute to the reliable diagnostics of schizophrenia at its early stages and to the plausible correction of cognitive impairments.

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Introduction: Common vervain is a plant used in traditional medicine. Its AE contains a vast number of compounds, hence its significant pharmacological potential. The monoamine hypothesis is the central theory of depression, and a majority of conventional antidepressants act on the monoaminergic system.

Methods: Experiments were conducted on Swiss albino sexually mature male mice. There were 6–8 animals in each of 5 subgroups (imipramine; fluoxetine; two different doses of AE – AE I, II; and VS). Forced Swimming Test (FST) and Tail Suspension Test (TST) were used to assess the antidepressive effect. Molecular docking experiments were performed using the program AutoDock 4.2, with 3D structures of crystalized proteins from the PDB database and 3D structures of ligands generated by the software Avogadro 2 0.8.0.
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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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. Malinic*, 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 expression of 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.