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# Expression of glutamatergic system genes in rats with stereotypies and audiogenic epilepsy (PM strain)

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*Motivation and Aim*: PM strain (an abbreviation for "pendulum-like movements") was obtained by selection for increased pendulum-like movements as a model of stereotypies in catatonic syndrome. In addition it has been noticed that PM rats also have a predisposition to seizures caused by audiogenic stimuli [1]. Considering the data on the involvement of the glutamatergic system in the etiopathogenesis of eplepsy and catatonia, the aim of the work was set: to study the expression of the glutamatergic system genes at the transcriptional level of the PM rats brain in comparison with Wistar controls.

*Methods and Algorithms*: Male rats of the PM and Wistar strains at the age of 6 months were used for the study. Using RT-qPCR *Grin1*, *Grin2A*, *Grin2B*, *Gria1*, *Grm2*, *Grm3*, *Grm5*, *Slc1a3*, *Slc1a2*, *Gad1*, *Gls*, *Slc17a6* mRNA level in brain structures (hippocampus, prefrontal cortex, striatum,) of PM and Wistar rats was estimated.

*Results*: The study revealed a high level of *Grm2* mRNA in hippocampus and prefrontal cortex of PM rats. This gene encodes a metabotropic autoreceptor, which, through a negative feedback mechanism, suppresses the release of glutamate into the synaptic cleft. In addition, in PM rats, an increase in the mRNA of another metabotropic receptor gene, *Grm5*, was shown in the prefrontal cortex. This postsynaptic receptor enhances glutamate flux through the NMDA receptors. There is evidence in the literature about the contribution of metabotropic receptors to the development of stress-related disorders [2]. Compared to the control, the level of mRNA of the *Slc1A3* gene of the EAAT1, responsible for the reuptake of glutamate, is higher in the hippocampus of PM rats, which may influence the amount of glutamate in the synaptic cleft. Data were obtained on a significant decrease in the hippocampus of PM rats in the mRNA of the *Gad1* gene, encoding the enzyme for the synthesis of GABA from glutamate. It is the imbalance of excitatory and inhibitory neurotransmitters that is believed to underlie psychopathological or neurological symptoms, including epilepsy [3].

*Conclusion*: The identified changes in the glutamate system genes expression in PM rats brain structures indicate changes in the neurotransmission of glutamate and can affect the level of excitation of these brain structures and electroconvulsive activity. The data obtained may help to identify the mechanisms of development of stereotypical pendulum-like movements and the susceptibility to audiogenic seizures of PM rats.

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