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THE EFFECT OF HISTONE DEACETYLASE INHIBITOR ON THE LEVELS OF GLUCOCORTICOID RECEPTOR EXPRESSION IN THE RAT FOREBRAIN UNDER HYPOXIA

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Activity of gene transcription depends to a large extent on the histone acetylation status and can be affected by different stimuli such as hypoxia. Histone deacetylase inhibitors promote gene transcription by facilitating histone acetylation and thus are considered as a new candidates for target therapy of post-hypoxic states. In the present study, using immunohistochemistry, the effects of the histone deacetylase inhibitor trihostatin A (TSA) on the levels of glucocorticoid receptor (GR) expression have been analyzed in the neocortex and hippocampus of rats in the original model of severe hypobaric hypoxia (SHH). It has been found that injections of TSA facilitated GR expression after SHH in the neocortex and CA1 field but not in the dentate girus of hippocampus of rats. Since GR overexpression is known to promote toxic action of the circulating glucocorticoid hormones on the hippocampal neurons, the stimulation of GR expression by TSA injections in the CA1 field of hippocampus may lead to maladaptation potentiating the detrimental impact of injurious factors such as hypoxia. The data obtained indicate that the therapy using histone deacetylase inhibitors may have unfavorable side effects for the vulnerable brain neurons.

K e y w o r d s: histone deacetylase inhibitors, histone acetylation, glucocorticoid receptors, hypoxia, brain neurons