SHORT COMMUNICATION

ENDOGENOUS LEVEL OF KYNURENIC ACID AND ACTIVITIES OF KYNURENINE AMINOTRANSFERASES FOLLOWING TRANSIENT GLOBAL ISCHEMIA IN THE GERBIL HIPPOCAMPUS

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Endogenous level of kynurenic acid and activities of kynurenine aminotransferases following transient global ischemia in the gerbil hippocampus.


The accumulated data indicate that massively released excitatory amino acids play a major role in mediating the acute ischemic neuronal degeneration. Kynurenic acid (KYNA), the endogenous glutamate receptor antagonist, displaying a particularly high affinity for the glycine-site of N-methyl-D-aspartate (NMDA) receptor, was shown to ameliorate ischemic brain damage and its altered metabolism was implicated in the pathogenesis of neurodegeneration during ischemia/anoxia. Thus, we investigated the effect of transient global ischemia in gerbils on the endogenous levels of KYNA and the activity of its biosynthethic enzymes, kynurenine aminotransferases I (KAT I) and II (KAT II) in the hippocampus, 24 and 72 h after the ischemic episode. The level of KYNA in CA1 area was not altered 24 and 72 h following transient global ischemia (39.7 ± 3.1 vs. 44.8 ± 4.2, and 46.3 ± 4.0 vs. 47.8 ± 3.9 fmol/mg of tissue). Similarly, the activities of KATs in CA1 area were not changed and reached 1.91 ± 0.11 vs. 1.8 ± 0.19 and 1.86 ± 0.1 vs. 1.7 ± 0.15 (KAT I), and 0.56 ± 0.2 vs. 0.43 ± 0.16 and 0.54 ± 0.08 vs. 0.55 ± 0.17 (KAT II) pmol KYNA/mg of tissue/h, respectively. The presented data indicate that KYNA production is preserved in CA1 area of gerbil hippocampus during early stages after ischemic insult.

Key words: hippocampus, CA1 area, kynurenic acid, kynurenine aminotransferases, transient global ischemia

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