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ABSTRACT NO. 26

TIME-DEPENDENT ALTERATION IN GAMMA-AMINOBUTYRIC ACID TRANSPORTER PROTEIN LEVELS DURING FERRIC CHLORIDE INDUCED EPILEPTOGENESIS

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Extracellular GABA concentration, which is a very important factor for explanation the failure of GABAergic inhibition, is regulated by specific Na^+-dependent transporter proteins on presynaptic terminals and glial cells. Recent molecular biological studies have revealed that at least three subtypes of GABA transporters, GAT-1, GAT-2 and GAT-3, exist in the rat brain, and the cDNA of each subtype has been isolated. We wonder if enhancement of glutamatergic excitatory synaptic transmission efficacy in the spontaneous seizure model is associated with changes in the levels of GABA transporter proteins. We performed a series of experiments utilizing quantitative Western blotting with antibodies specific to the individual GABA transporters, GAT-1 and GAT-3, to test this hypothesis. To investigate the role of hippocampal GABA transporters following epileptogenesis induced by ferric cation injection into amygdaloid body, we examined alterations in the expression of hippocampal GAT-1, GAT-2 and GAT-3 GABA transporter proteins using Western blot. Following bilateral increase (about 150-250%) at 5-15days after injection, GAT-1 and GAT-2 proteins came to control levels at 30 days later bilaterally. In contrast, GAT-3 protein was also significantly increased bilaterally at 5-15 days, and continued high level bilaterally at 30 days after injection. Since alterations in GAT-1 returned to control levels at 30 days, the increases in GAT-1 seem to be transient responses to seizure activity. GAT-3 subtype transporter, which continued to be at high levels in the chronic phase, is mainly involved in seizure activity in following epileptogenesis induced by ferric ion injected into the amygdaloid body.