

Prevention of epileptic seizures by taurine.

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Parenteral injection of kainic acid (KA), a glutamate receptor agonist, causes severe and stereotyped behavioral convulsions in mice and is used as a rodent model for human temporal lobe epilepsy. The goal of this study is to examine the potential anti-convulsive effects of the neuro-active amino acid taurine, in the mouse model of KA-induced limbic seizures. We found that taurine (43 mg/Kg, s.c.) had a significant antiepileptic effect when injected 10 min prior to KA. Acute injection of taurine increased the onset latency and reduced the occurrence of tonic seizures. Taurine also reduced the duration of tonic-clonic convulsions and mortality rate following KA-induced seizures. Furthermore, taurine significantly reduced neuronal cell death in the CA3 region of the hippocampus, the most susceptible region to KA in the limbic system. On the other hand, supplementation of taurine in drinking water (0.05%) for 4 continuous weeks failed to decrease the number or latency of partial or tonic-clonic seizures. To the contrary, we found that taurine-fed mice showed increased susceptibility to KA-induced seizures, as demonstrated by a decreased latency for clonic seizures, an increased incidence and duration of tonic-clonic seizures, increased neuronal death in the CA3 region of the hippocampus and a higher post-seizure mortality of the animals. We suggest that the reduced susceptibility to KA-induced seizures in taurine-injected mice is due to an increase in GABA receptor function in the brain which increases the inhibitory drive within the limbic system. This is supported by our in vitro data obtained in primary neuronal cultures showing that taurine acts as a low affinity agonist for GABA(A) receptors, protects neurons against kainate excitotoxic insults and modulates calcium homeostasis. Therefore, taurine is potentially capable of treating seizure-associated brain damage.

PMID: 12908638 [PubMed - indexed for MEDLINE]