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Taurine attenuates D-[3H]aspartate release evoked by depolarization in ischemic corticostriatal slices.

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Abstract

Taurine is thought to be protective in ischemia due to its neuroinhibitory effects. The present aim was to assess the ability of taurine to attenuate glutamate release evoked by ischemia and to determine which component of this release is affected. The release of preloaded D-[(3)H]aspartate (a non-metabolized analog of glutamate) from superfused murine corticostriatal slices was used as index of glutamate release. Preincubation of corticostriatal slices with 10 mM taurine reduced the D-[(3)H]aspartate release evoked by either chemical ischemia (0.5 mM NaCN in glucose-free medium) or oxygen-glucose deprivation. The taurine uptake inhibitor guanidinoethanesulfonate (5 mM), the glycine receptor antagonist strychnine (0.1 mM) and the GABA(A) receptor antagonist bicuculline (0.1 mM) did not block the taurine effect. To determine which component of ischemia-induced glutamate release is affected by taurine, three pathways of this release were pharmacologically modeled. Unlabeled D-aspartate (0.5 mM) and hypo-osmotic medium (NaCl reduced by 50 mM) evoked D-[(3)H]aspartate release via homoexchange and hypo-osmotic release pathways, respectively. Taurine did not influence these pathways. However, it suppressed the synaptic release of D-[(3)H]aspartate evoked by the voltage-gated sodium channel opener veratridine (0.1 mM). Taurine thus reduces glutamate release under ischemic conditions by affecting the depolarization-evoked component.

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