Sulforaphane is anticonvulsant and improves mitochondrial function.

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Resumen

The nuclear factor erythroid 2-related factor 2 pathway (Nrf2) has been previously identified to protect the brain against various impacts. Here, we investigated the effect of the Nrf2 activator sulforaphane in various seizure models and hippocampal mitochondria' bioenergetics. We found that daily injections of sulforaphane for 5 days elevated the seizure thresholds to 6 Hz stimulation and fluorothyl-, but not pentylenetetrazole-induced tonic seizures and protected mice against pilocarpine-induced status epilepticus (SE). Also, sulforaphane increased the antioxidant defences within hippocampal formations and blood plasma. In addition, sulforaphane treatment reduced the extent of hippocampal lipid peroxidation 24 h post-SE and protected hippocampal mitochondria against SE-induced reduction in state 2 and uncoupler-stimulated state 3 respiration. SE-mediated partial loss of rotenone-sensitive and complex II-driven respiration was reduced, consistent with the enhanced activities of complexes I and II in sulforaphane-related SE mice. In mitochondria isolated from both no SE and SE mice, sulforaphane increased state 3 respiration and respiration linked to ATP synthesis, which may contribute to its anticonvulsant and antioxidant effects by providing more ATP for cellular vital and protective functions. However, sulforaphane did not prevent SE-induced hippocampal cell death. In conclusion, sulforaphane and/or Nrf2 activation are viable anticonvulsant strategies, which are antioxidant and enhance mitochondria' function, especially the ability to produce ATP.

Palabras clave

Palabras clave de autor:epilepsy; mitochondrial respiration; Nrf2; pilocarpine; seizure; sulforaphane
KeyWords Plus:MOUSE PILOCARPINE MODEL; TEMPORAL-LOBE EPILEPSY; TRAUMATIC BRAIN-INJURY; OXIDATIVE STRESS; KETOGENIC DIET; INTRACEREBRAL HEMORRHAGE; ANTIEPILEPTIC DRUGS; SKELETAL-MUSCLE; SEIZURE MODELS; NRF2

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