Role of JNK isoforms in the kainic acid experimental model of epilepsy and neurodegeneration

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Chemoconvulsants that induce status epilepticus in rodents have been widely used over the past decades due to their capacity to reproduce with high similarity neuropathological and electroencephalographic features observed in patients with temporal lobe epilepsy (TLE). Kainic acid is one of the most used chemoconvulsants in experimental models. KA administration mainly induces neuronal loss in the hippocampus. We focused the present review inthe c-Jun N-terminal kinase-signaling pathway (JNK), since it has been shown to play a key role in the process of neuronal death following KA activation. Among the three isoforms of JNK (JNK1, JNK2, JNK3), JNK3 is widely localized in the majority of areas of the hippocampus, whereas JNK1 levels are located exclusively in the CA3 and CA4 areas and in dentate gyrus. Disruption of the gene encoding JNK3 in mice renders neuroprotection to KA, since these animals showed a reduction in seizure activity and a diminution in hippocampal neuronal apoptosis. In light of this, JNK3 could be a promising subcellular target for future therapeutic interventions in epilepsy.

Apoptosis

C-Jun N-terminal kinase signaling pathway

HippocampuS

Kainic acid

Neuroprotection

Review

anticonvulsive agent

isoenzyme

kainic acid

mitogen activated protein kinase 10

signal transducing adaptor protein

stress activated protein kinase

animal

chemically induced

deficiency

disease model

drug effects

enzymology

epilepsy

genetics

hippocampus

human

knockout mouse

MAPK signaling

metabolism

mouse

nerve degeneration

pathology

pathophysiology

Adaptor Proteins, Signal Transducing

Animals

Anticonvulsants

Disease Models, Animal

Epilepsy

Hippocampus

Humans

Isoenzymes

JNK Mitogen-Activated Protein Kinases

Kainic Acid

MAP Kinase Signaling System

Mice

Mice, Knockout

Mitogen-Activated Protein Kinase 10

Nerve Degeneration