Neuroprotection in hypoxic-ischemic brain injury targeting glial cells

Mucci S.

Herrera M.I.

Barreto G.E.

Kolliker-Frers R.

Capani F.

Brain injury constitutes a disabling health condition of several etiologies. One of the major causes of brain injury is hypoxia-ischemia. Until recently, pharmacological treatments were solely focused on neurons. In the last decades, glial cells started to be considered as alternative targets for neuroprotection. Novel treatments for hypoxia-ischemia intend to modulate reactive forms of glial cells, and/or potentiate their recovery response. In this review, we summarize these neuroprotective strategies in hypoxia-ischemia and discuss their mechanisms of action. © 2017 Bentham Science Publishers.

Brain injury

Glial cells

Hypoxia-ischemia

Neuroprotection

Pharmacological treatments

AMPA receptor

AMPA receptor antagonist

apocynin

bungarotoxin receptor

cannabinoid 2 receptor agonist

carnosine

catalpol

cholinergic receptor stimulating agent

cynarine

deferoxamine

diclofenac

enkephalin

ephrin A3

ephrin receptor A4

fingolimod

ginsenoside Rb 1

glutamic acid

hypophysis adenylate cyclase activating polypeptide

interleukin 4

kainic acid receptor antagonist

mercaptamine

microRNA

microRNA 181

neuroprotective agent

o 1966

propofol

small interfering RNA

sphingosine kinase 1

sulforedoxin

unclassified drug

unindexed drug

neuroprotective agent

astrocyte

cellular immunity

drug conjugation

drug design

drug mechanism

drug targeting

gene expression

gene expression regulation

glia cell

human

hypothermia

hypoxic ischemic encephalopathy

microglia

molecularly targeted therapy

neuromodulation

neuroprotection

nonhuman

priority journal

receptor blocking

Review

stem cell

animal

drug delivery system

drug effect

glia

hypoxic ischemic encephalopathy

metabolism

nerve cell

neuroprotection

pathology

physiology

trends

Animals

Drug Delivery Systems

Humans

Hypoxia-Ischemia, Brain

Neuroglia

Neurons

Neuroprotection

Neuroprotective Agents