Neuroprotective Agent

- Therapeutic Potential of Ellagic Acid in Liver Diseases
- Advances in the Exploration of Coordination Complexes of Vanadium in the Realm of Alzheimer's Disease: A Mini Review
- Withaferin A Rescues Brain Network Dysfunction and Cognitive Deficits in a Mouse Model of Alzheimer's Disease
- Brain Neurotrophins and Plant Polyphenols: A Powerful Connection
- Successful salvage therapy of ruxolitinib on interstitial pneumonia after long COVID or post-COVID-19 syndrome with follicular lymphoma: two case reports and literature review
- Nerve Growth Factor in Pediatric Brain Injury: From Bench to Bedside
- Proinflammatory cytokines - as a marker of the effectiveness of neuroprotection in children with perinatal nervous system damage
- Piper Kadsura Extract and its Bioactive Components Attenuates Aβ₂₅₋₃₅-Induced Neurotoxicity by Modulating Glycolysis and Activating the SIRT1/PPARγ/GLUT1 Axis

A cerebroprotective agent is a substance or compound that can protect the brain from damage due to various factors such as injury, disease, or toxins. The term “cerebro” refers to the brain, and “protective” means to safeguard or defend.

There are various types of cerebroprotective agents, including natural compounds found in plants, as well as synthetic drugs. These agents may act through different mechanisms, such as reducing inflammation, increasing blood flow to the brain, scavenging free radicals, and preventing cell death.

Some examples of cerebroprotective agents include:

Antioxidants: Substances that can neutralize free radicals and reduce oxidative stress, which can lead to brain damage. Examples include vitamins C and E, beta-carotene, and flavonoids.

Nootropics: Also known as “smart drugs,” these substances can enhance cognitive function and protect the brain from damage. Examples include piracetam, aniracetam, and modafinil.

Neurotrophic factors: Proteins that can promote the growth and survival of neurons in the brain. Examples include brain-derived neurotrophic factor (BDNF) and nerve growth factor (NGF).

Anti-inflammatory agents: Substances that can reduce inflammation in the brain, which can contribute to brain damage. Examples include nonsteroidal anti-inflammatory drugs (NSAIDs), such as aspirin and ibuprofen.

When treating acute ischemic stroke patients in our daily clinical practice, we strive to achieve recanalization of the occluded blood vessel as fast as possible using pharmacological thrombolysis and mechanical clot removal. However, successful recanalization does not equal successful reperfusion of the ischemic tissue due to mechanisms such as microvascular obstruction. Even if successful reperfusion is achieved, numerous other post-recanalization tissue damage mechanisms may impair patient outcomes, namely blood-brain barrier breakdown, reperfusion injury and excitotoxicity, late secondary changes, and post-infarction local and global brain atrophy. Several cerebroprotectants are currently evaluated as adjunctive treatments to pharmacological thrombolysis and mechanical clot removal, many of which interfere with post-recanalization tissue damage

pathways. However, our current lack of knowledge about the prevalence and importance of the various post-recanalization tissue damage mechanisms makes it difficult to reliably identify the most promising cerebroprotectants and design appropriate clinical trials to evaluate them. Serial human MRI studies with complementary animal studies in higher-order primates could provide answers to these critical questions and should be first conducted to allow for adequate neuroprotection trial design, which could accelerate the translation of cerebroprotective agents from bench to bedside to further improve patient outcomes [1].

Nimodipine

The mechanism of the protective competence of nimodipine is unknown. Therefore, a study, we established an in vitro model to examine the survival of Neuro2a cells after different stress stimuli occurring during surgery with or without nimodipine. Nimodipine significantly decreased ethanol-induced cell death of cells up to approximately 9% in all tested concentrations. Heat-induced cell death was diminished by approximately 2.5% by nimodipine. Cell death induced by mechanical treatment was reduced up to 15% by nimodipine. These findings indicate that nimodipine rescues Neuro2a cells faintly, but significantly, from ethanol-, heat- and mechanically-induced cell death to different extents in a dosage-dependent manner. This model seems suitable for further investigation of the molecular mechanisms involved in the neuroprotective signal pathways influenced by nimodipine [2].

Melatonin appears to have neuroprotective effects on the secondary brain damage while nimodipine and nimodipine plus melatonin combination did not show such neuro-protective effects on the secondary brain injury [3].

Methoxyflurane

see Methoxyflurane.

Erythropoietin and curcumin showed promising neuroprotective effects in various models of Alzheimer's dementia.

Citicoline

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1)
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2)
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3)
Ismailoglu O, Atilla P, Palaoglu S, Cakar N, Yasar U, Kilinc K, Kaptanoglu E. The therapeutic effects of melatonin and nimodipine in rats after cerebral cortical injury. Turk Neurosurg. 2012;22(6):740-6. doi:

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