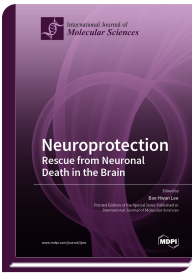
International Journal of
Molecular Sciences

IMPACT FACTOR 4.9
Indexed in: PubMed
CITESCORE 8.1

Special Issue Reprint

Neuroprotection: Rescue from Neuronal Death in the Brain



www.mdpi.com/books/reprint/4368

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ISBN 978-3-0365-1994-4 (Hardback)
ISBN 978-3-0365-1995-1 (PDF)

The brain is vulnerable to injury. Following injury in the brain, apoptosis or necrosis may occur easily, leading to various functional disabilities. Neuronal death is associated with a number of neurological disorders including hypoxic ischemia, epileptic seizures, and neurodegenerative diseases.

The brain subjected to injury is regarded to be responsible for the alterations in neurotransmission processes, resulting in functional changes. Oxidative stress produced by reactive oxygen species has been shown to be related to the death of neurons in traumatic injury, stroke, and neurodegenerative diseases. Therefore, scavenging or decreasing free radicals may be crucial for preventing neural tissues from harmful adversities in the brain. Neurotrophic factors, bioactive compounds, dietary nutrients, or cell engineering may ameliorate the pathological processes related to neuronal death or neurodegeneration and appear beneficial for improving neuroprotection. As a result of neuronal death or neuroprotection, the brain undergoes activity-dependent long-lasting changes in synaptic transmission, which is also known as functional plasticity.

Neuroprotection implying the rescue from neuronal death is now becoming one of global health concerns. This Special Issue attempts to explore the recent advances in neuroprotection related to the brain. This Special Issue welcomes original research or review papers demonstrating the mechanisms of neuroprotection against brain injury using in vivo or in vitro models of animals as well as in clinical settings. The issues in a paper should be ufficient data or evidence.

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