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Molecular Research and Therapeutic Targets in Acute Settings: Ischemic and Traumatic Brain Injuries

Guest Editor:

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Message from the Guest Editor

Dear Colleagues,

The pathophysiology of cerebral ischemia and brain trauma indicates that similar mechanisms contribute to the loss of cellular integrity and tissue damage.

Brain injuries result from the complex interplay of multiple pathways including excitotoxicity, ionic imbalance, oxidative/nitrative stress and blood-brain barrier (BBB) disruption. With cell type-specific pathomechanisms, acute cerebral insults activate neuronal cell death, inflammation from microglia activation and immune cell infiltration, accompanied with astrogliosis, and complex glia-neuron exchanges, and they are important elements in the pathological onset and the progression of ischemic or trauma-triggered brain damage.

From exploring these molecular mechanisms and dissecting the role of specific cell-type signaling pathways or cell–cell interactions, each contribution will aim to provide insights that pave the way for more effective therapeutic approaches.

I look forward to receiving your contributions.



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Editor-in-Chief

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Message from the Editor-in-Chief

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