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Oxidative Stress and Proteinopathy in Alzheimer's Disease

Guest Editor:

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Deadline for manuscript submissions:
closed (30 September 2024)

Message from the Guest Editor

Dear Colleagues,<false,>The multifactorial nature of Alzheimer's disease (AD) has given rise to several research debates on the early events that led to AD. Of note, interrogation of post-mortem AD brain and several neuroimaging studies has revealed accumulation and misfolding of certain proteins (prominantely amyloid beta and tau) as the important primary event playing a significant role in the initiation and progression of the disease. This is of particular relevance since this results in synaptic dysfunction and loss, leading to cognitive decline and ultimately results in neuronal cell death. An intriguing and complex relationship has been shown between Aβ and mitochondrial dysfunction, redox metal imbalance, failure of protein clearance mechanism, advanced glycation end-products, and calcium excitotoxicity that may further contribute to the oxidative stress in the brains of AD patients. Understanding these interactions may thus aid in the identification of potential neuroprotective therapies that could be clinically useful.

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Message from the Editor-in-Chief

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