Special Issue

□-Synuclein in Neurons and Glia: From Physiology to Pathology

Message from the Guest Editors

The physiological role of the protein □ -synuclein is debated; it is expressed constitutively in neurons and found in glia under certain pathological conditions. □-Synuclein fibrillary aggregation and spreading through neuroanatomically connected regions is a hallmark of a series of neuropathologies termed synucleinopathies that include Parkinson 's disease, dementia with Lewy bodies, pure autonomic failure, and multiple system atrophy. However, several questions remain unanswered: is □-synuclein aggregation a cause or effect of the pathological process? What determines the transition from the physiological to pathological state of □-synuclein? Is neuronal □-synuclein different from the □-synuclein found in glial cells? Is it worth using □-synuclein in animal models? In this Special Issue, we want to shed light upon the complex scenario of □-synuclein transition from physiology to pathology in synucleinopathies, with a wide view from the clinic to the basic experimental knowledge.

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Deadline for manuscript submissions

closed (31 December 2021)

International Journal of Molecular Sciences

an Open Access Journal by MDPI

Impact Factor 4.9
CiteScore 9.0
Indexed in PubMed

mdpi.com/si/51748

International Journal of Molecular Sciences
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The International Journal of Molecular Sciences (*IJMS*, ISSN 1422-0067) is an open access journal, which was established in 2000. The journal aims to provide a forum for scholarly research on a range of topics, including biochemistry, molecular and cell biology, molecular biophysics, molecular medicine, and all aspects of molecular research in chemistry. *IJMS* publishes both original research and review articles, and regularly publishes special issues to highlight advances at the cutting edge of research. We invite you to read recent articles published in *IJMS* and consider publishing your next paper with us.

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