



Molecular Mechanisms of Alpha-Synuclein in Parkinson's Disease and Other Synucleinopathies

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Message from the Guest Editors

Dear Colleagues,

Twenty-five years ago, researchers discovered that mutations in the *alpha-synuclein* (*SNCA* or *PARK1*) gene are responsible for an autosomal dominant form of Parkinson's disease (PD), and that alpha-synuclein protein is an important component of Lewy bodies (the hallmark of PD). Thereafter, it was discovered that alpha-synuclein is accumulated in Lewy bodies in other disorders such as PD with dementia (PDD) and dementia with Lewy bodies (DLB), and in the form of glial cytoplasmic inclusions in multiple-system atrophy (MSA). The term "synucleinopathies" was coined to encompass PD, PDD, DLB, and MSA. Alpha-synuclein is expressed not only in the central nervous system (CNS), but also in some peripheral diseases, and is a reliable biological marker for these diseases. Since 1997, approximately 12,000 articles have been published regarding alpha-synuclein, but the role of this protein in PD and other synucleinopathies is not yet fully understood. For this reason, we propose a Special Issue on the molecular mechanisms of alpha-synuclein in PD and other synucleinopathies and welcome submissions on this this topic.





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