Special Issue

Exploring Tauopathy Through a Molecular Lens for Comprehensive Understanding

Message from the Guest Editors

Tauopathies, which involve dysregulation of the essential neuronal microtubule-associated protein Tau, are the most widespread neurodegenerative dementias. Unlike the familial frontotemporal dementia with Parkinsonism on 17 (FTDP-17), involving mutations in the MAPT gene, other Tauopathies are characterized as a wild type, hyperphosphorylated Tau isoforms that underlie neuronal dysfunction and neurotoxicity. However, the mechanisms that trigger the transformation of physiological Tau isoforms into hyperphosphorylated soluble or aggregated species are not well understood but are at the heart of Tau-dependent pathogeneses. For diagnoses and therapies, the critical molecular mechanisms linked with and ostensibly permissive to the progression of Tauopathies from nearly early non-symptomatic to the catastrophic consequences of dementia, also remain mostly undefined. This Special Issue will focus on the molecular mechanisms of Tauopathies, physiological functions of Tau, uncovering the unique function of each isoform, Tau-related molecular mechanisms that cause early cognitive and synaptic impairments and mechanisms that promote physiological Tau converts to pathological Tau.

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Deadline for manuscript submissions

closed (15 March 2025)



Cells

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Impact Factor 5.2 CiteScore 10.5 Indexed in PubMed



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Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. Cells encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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