Special Issue

Advances in Tau Protein Research

Message from the Guest Editor

Alzheimer's disease is the most common dementia, affecting over 57 million people worldwide. Neurofibrillary tangles were described over 100 years ago, but only in the 1980s were they identified as amyloid filaments of hyperphosphorylated tau. These tau filaments, with distinct ultrastructures, are key hallmarks of tauopathies. In the 1990s, mutations in the MAPT gene encoding tau were linked to frontotemporal dementia and parkinsonism, suggesting tau filament formation is sufficient to cause neurodegeneration. The idea that pathological tau spreads between cells arises from its stepwise brain deposition and experimental support for its prion-like behavior. This Special Issue aims to advance our understanding of the physiological and pathological mechanisms around tau protein, including, among others, the interactions with cellular factors, emerging therapeutic strategies, molecular mechanisms of templated seeded aggregation, and toxic functions of protein aggregates. Studies providing such information are welcomed and will help elucidate the molecular basis for designing new treatments as well as deeper understanding of protein aggregation pathologies.

Guest Editor

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