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Protein Post-Translational Modifications and Protein Aaggregation in Neurodegenerative Diseases

Guest Editor:

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Deadline for manuscript submissions:
closed (25 March 2020)

Message from the Guest Editor

Dear Colleagues,

Undesired or aberrant protein aggregation is common to a number of neurodegenerative diseases, with accumulation of aggregated proteins thought to be neurotoxic. Examples include: Aβ or tau protein aggregates in Alzheimer's disease; α-synuclein deposits within Lewy bodies (LBs) in Parkinson's disease, multiple system atrophy, and dementia with LBs; and poly-glutamine tracts in Huntington's disease. A growing area of research in neurodegenerative disease focusses upon how PTMs promote protein aggregation, and how modified and aggregated proteins may be resistant to degradation or removal by cellular machinery including proteasomal or autophagic mechanisms. The importance of protein PTMs and associated protein aggregation are underscored by current clinical research to develop drugs or biological therapies with anti-(protein)aggregation properties. This special issue will broadly cover PTMs that may influence protein aggregation, methods to detect aggregated proteins, and the relationship between protein aggregation and the pathogenesis or progression of neurodegenerative diseases.

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Message from the Editor-in-Chief

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