



Protein Misfolding in Neurodegenerative Diseases: Recent Advances and Therapeutical Implications

Guest Editor:

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Message from the Guest Editor

Although the precise mechanisms by which protein misfolding and aggregation is involved in neurodegeneration are still unclear, compelling evidence supports a loss of physiological function and/or gain of toxic function upon protein misfolding to a major role in the pathogenesis of protein conformational diseases. Building on these findings, several therapeutical approaches have been proposed, which target the different steps in the synthesis and processing of aggregation-prone proteins, including i) lowering the concentration of the amyloidogenic protein, ii) stabilization of the protein native state conformation, iii) modulation of the proteome quality control via proteasome and autophagy pathways, and iv) decreasing the vicious cycle of amyloid formation seeding and spreading. The aim of this Special Issue is to summarise the current knowledge on the molecular mechanisms underlying protein misfolding, and to highlight emerging therapeutic strategies for the prevention or treatment of neurodegenerative disorders associated with protein aggregation.





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