Special Issue

Non-Antibody Based Anti-Aggregation Therapies Targeting Amyloid-Beta, Tau and Alpha-Synuclein and Other Proteins Associated With Neurodegenerative Disorders

Message from the Guest Editor

Protein aggregation is a hallmark of neurodegenerative disorders, including Parkinson's (PD), Alzheimer's (AD), Huntington's (HD), frontotemporal lobar degeneration (FTLD), amyotrophic lateral sclerosis (ALS), and others. These diseases often involve multiple aggregating proteins, such as AX, Tau, and X-Syn, which can exhibit prion-like propagation, spreading pathology throughout the brain. Current treatments, like anti-A antibodies (lecanemab, donanemab), show limited efficacy, requiring frequent IV infusions and carrying risks such as brain swelling. Their high cost and narrow targeting (e.g., only A underscore the need for alternative therapies, including small molecules, peptides, and antisense oligonucleotides, to broadly combat protein aggregation. This Special Issue seeks research on nonantibody therapeutics targeting AX, Tau, X-Syn, and other aggregation-prone proteins (e.g., TDP-43, SOD1, HTT, PrPc) to advance treatment options for neurodegenerative diseases.

Guest Editor

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Editor-in-Chief

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