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 A□, Tau, and □-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 non-antibody therapeutics targeting A□, Tau, □-Syn, and other aggregation-prone proteins (e.g., TDP-43, SOD1, HTT, PrP^{c}) to advance treatment options for neurodegenerative diseases.

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