MOLECULAR NEURODEGENERATION

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# Prion-like aspects of cerebral amyloidosis

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The commonality of many neurodegenerative disorders is the predictable temporal occurrence and progression of specific aggregated proteins in the brain. The hallmark proteopathy is Alzheimer's disease in which aggregated amyloid-β peptide (Aβ) is deposited in the brain parenchyma as amyloid plaques. Multiple evidence suggests that β-amyloidosis is induced by aggregated Aβ which can spread within and among brain regions and act as corruptive templates (seeds) that induce a chain-reaction of Aβ misfolding and aggregation. The insight that the prion paradigm may also apply to cerebral β-amyloidosis and other proteopathies suggests new avenues in search of biomarkers and novel therapeutic strategies.

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