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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 ($A\beta$) is deposited in the brain parenchyma as amyloid plaques. Multiple evidence suggests that β -amyloidosis is induced by aggregated $A\beta$ which can spread within and among brain regions and act as corruptive templates (seeds) that induce a chain-reaction of $A\beta$ 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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