PRION-LIKE SPREADING IS A RELEVANT THEORY FOR ALL NEURODEGENERATIVE DISEASES: NO

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As of now, there is no adequate explanation for the orderly progression of the pathological changes in Parkinson's disease (PD), which were described by Braak *et al*. These changes are characterized by Lewy bodies and neurites, which are composed primarily by α-synuclein (AS). AS are normal membrane proteins, the functions of which are still unknown.

Prions are also membrane proteins of unknown function. Natively, prions occur as α-sheet but can assume β-sheet conformation, which are assumed to be neurotoxic, and accelerate conversion of further prion molecules to the abnormal form.

It has been hypothesized that AS can also assume a toxic structure, fibrillar AS, which causes damage to neurons and later are shed to the neuropil and are taken up by neighboring dendrites and neurons, to further the spread of damage. This suggested mechanism is quite unlike that of prion toxicity and several of its assumed steps need to be examined carefully.