PARKINSON'S DISEASE IS A PRION DISORDER - YES

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Prions are infectious agents that are comprised solely of misfolded proteins characterized by a propensity to misfold to form toxic oligomers, to polymerize to generate amyloid plaques, to cause neurodegeneration and to spread to affect neighboring regions in an infectious manner. Creutzfeldt-Jacob disease, Kuru, Gerstmann–Sträussler–Scheinker (GSS) syndrome, and fatal familial insomnia are examples of human prion diseases. A body of evidence now suggests that Parkinson's disease (PD) is also a prion disorder, and α-synuclein a prion.

Mutations in α-synuclein are associated with a rare, autosomal dominant hereditary form of PD, and α-synuclein is now known to be a major component of the Lewy bodies and Lewy neurites that characterize the pathology of the sporadic form of the disease. It is also now known that duplication or triplication of the wildtype α-synuclein gene can cause a familial form of PD, suggesting that increased levels of the protein itself can cause PD. Importantly, Lewy pathology has now been discovered in embryonic dopamine neurons that were implanted into the striatum of PD patients, suggesting transfer from affected to unaffected dopamine neurons. Based on these observations we raised the possibility that PD a prion disorder. Considerable laboratory evidence now supports this hypothesis. α-Synuclein uptake by healthy neurons has been demonstrated both "in vitro" and "in vivo". The intracerebral injection of purified α-synuclein fibrils leads to α-synuclein aggregates in host neurons with neurodegeneration, motor dysfunction, and extension of pathology to anatomically-related neurons. Importantly, these findings do not occur in α-synuclein null animals consistent with α-synuclein aggregates acting as a template to promote misfolding of host α-synuclein. Further, inoculates derived from the brains of older transgenic rodents that overexpress α-synuclein accelerate the onset of the illness when injected into the brains of young transgenic animals. More recently, it has been shown that inoculates derived from Lewy pathology in patients with multiple system atrophy or PD induced widespread α-synuclein aggregates throughout the hemisphere and brain stem and clinical dysfunction in rodents and primates. Collectively, these studies strongly support the likelihood, that α-synuclein is a prion, and PD is a prion disorder.