

Parkinson's disease: a puzzling widespread distribution of pathological changes

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Parkinson's disease (PD) is the most common motor disorder and the second most common neurodegenerative disease after Alzheimer's disease. The major neuropathological features of Parkinson's disease are abnormally misfolded intracellular protein aggregates, referred to as Lewy bodies and Lewy neurites, which consist largely of insoluble deposits of the alpha-synuclein protein. Currently, the etiology and pathogenesis of the neurodegenerative process in Parkinson's disease are only marginally known and, as of yet, no single diagnostic or prognostic biomarker has been identified.

Unlike many other neurodegenerative diseases, PD-associated pathology occurs not only in the central, but also in the peripheral nervous system. It is currently presumed that the mechanisms by which a pathological alpha-synuclein spreads throughout the nervous system and triggers neurodegeneration are very similar to those of prion diseases. This means that the pathologically altered or misfolded alpha synuclein may be transmitted through a chain of interconnected neurons in a prion-like manner and induces misfolding of the native alpha synuclein of the newly affected neurons. Whether the propagation of the pathological process is bi-directional or uni-directional from the central to the peripheral nervous system or vice versa is currently unknown. This talk will discuss the spectrum of pathological changes in PD and its clinical implications.