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Molecular Pathogenesis and Neuroinflammation in Parkinson Disease beyond Alpha-synuclein

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Various mechanisms play an essential role in the pathogenesis of Parkinson's disease, including a disruption of the cellular energy balance (mitochondrial dysfunction) and oxidative stress and disruption of protein breakdown (lysosomal and proteasomal dysfunction). The protein α -synuclein plays a central role in the pathogenesis of Parkinson's disease and is involved in many intracellular functions or their disruption in Parkinson's disease. A number of important cellular processes are inhibited by aggregated α -synuclein. The disruption of these processes, in turn, leads to increased aggregation of α -synuclein. Therefore, protein misfolding, aggregation, and accumulation of aggregated α -synuclein is a key feature of Parkinson's disease. In the course of the disease, there is an inflammatory reaction in the brain (neuroinflammation), in which mainly microglial cells are involved. The exact triggers of neuroinflammation are not known. However, it is known that aggregated α -synuclein and neuromelanin are able to activate microglia. In the course of neuroinflammation, there is a change in the expression of toll-like receptors (TLR) on the microglial cells. Hence, in Parkinson's disease, both the aggregation of proteins, primarily α -synuclein and one that occurs during the course of the disease play a role in neuroinflammation with microglial activation in the brain of Parkinson's patients playing a role in the pathogenesis of the disease.

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