Neuronal Parkinson inclusions are different than expected

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Résumé

Parkinson's disease (PD) is a neurodegenerative disease of multifactorial origins. So far, available treatments only affect symptoms. The molecular causes or cellular mechanisms in PD are not understood, reliable diagnosis in patients is difficult, and no treatment is available to slow down or prevent progression of the disease. The protein alpha-synuclein is a prime suspect for the cause of Parkinson's disease. This soluble protein is capable of aggregating into prionoid fibrils in a variety of conformational strains. The structural analysis of these via cryo-Electron Microscopy (cryo-EM) will be presented, and high-resolution structures of different alpha-synuclein fibril strains from wild-type, as well as post-translationally modified, truncated, or PD-relevant mutation carrying protein will be presented and discussed. In parallel, electron microscopy studies of post-mortem human brain from Parkinson's disease tissue donors with a post-mortem delay as short as 3 hours will be presented, and the ultrastructure of Lewy body plaques in the brain tissue, which are highly enriched in the protein alpha-synuclein, will be discussed. These studies have implications for our understanding of the possible causes and mechanisms of Parkinson's disease.

Mots-Clés: STED, microscopy, Parkinson's disease

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