

Animal models are useful in understanding PD pathogenesis

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Parkinson's disease (PD) is the second most common neurodegenerative disease after Alzheimer's disease. Nigrostriatal dopaminergic neurodegeneration is shared with other parkinsonian disorders: including some genetic forms of Parkinsonism, but many of these disorders do not have Lewy bodies. An ideal animal model of PD should exhibit age-dependent and progressive dopaminergic neurodegeneration, motor dysfunction, and abnormal alpha-synuclein pathology. Mitochondrial oxidative phosphorylation, autophagy-lysosomal metabolism, ubiquitin-proteasome protein degradation, and endoplasmic reticulum stress/unfolded protein response are impaired cellular functions in PD (Jiang and Dickson 2018). Many rodent models have been developed to investigate PD using genetic or toxin based strategies, but all have significant limitations. On the other hand there are several promising therapeutic agents for the treatment of PD. Therefore we need much better models of the disease (Majláth et al. 2016, Török et al. 2016, Chen et al. 2017, Tronci and Francado 2018, Francado 2018).