Glucagon-like peptide-1 receptor (GLP1R) agonist has gained interest as a potential treatment for Parkinson's disease (PD). However, the exact mechanisms responsible for the therapeutic effects of GLP-1R-related agonists are not yet fully understood.

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Wang et al. explores the effects of early treatment with PT320, a sustained release formulation of the GLP-1R agonist Exenatide, on mitochondrial functions and morphology in a progressive PD mouse model, the MitoPark (MP) mouse.

The findings demonstrate that administration of a clinically translatable dose of PT320 ameliorates the reduction in tyrosine hydroxylase expression, lowers reactive oxygen species (ROS) levels, and inhibits mitochondrial cytochrome c release during nigrostriatal dopaminergic denervation in MP mice. PT320 treatment significantly preserved mitochondrial function and morphology but did not influence the reduction in mitochondria numbers during PD progression in MP mice. Genetic analysis indicated that the cytoprotective effect of PT320 is attributed to a reduction in the expression of mitochondrial fission protein 1 (Fis1) and an increase in the expression of optic atrophy type 1 (Opa1), which is known to play a role in maintaining mitochondrial homeostasis and decreasing cytochrome c release through remodeling of the cristae.

The findings suggest that the early administration of PT320 shows potential as a neuroprotective treatment for PD, as it can preserve mitochondrial function. Through enhancing mitochondrial health by regulating Opa1 and Fis1, PT320 presents a new neuroprotective therapy in PD¹⁾

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Wang V, Tseng KY, Kuo TT, Huang EY, Lan KL, Chen ZR, Ma KH, Greig NH, Jung J, Choi HI, Olson L, Hoffer BJ, Chen YH. Attenuating mitochondrial dysfunction and morphological disruption with PT320 delays dopamine degeneration in MitoPark mice. J Biomed Sci. 2024 Apr 17;31(1):38. doi: 10.1186/s12929-024-01025-6. PMID: 38627765.

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