

# Aminochrome as New Preclinical Model to Find New Pharmacological Treatment that Stop the Development of Parkinson's Disease

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## Resumen

The pharmacological treatment of Parkinson's disease (PD) is limited to dopamine agonists and anti-cholinergic drugs that do not stop the progress of disease. L-Dopa was introduced to the treatment in 1967; this drug is still the best and most commonly used drug since it generates a real improvement in patient quality of life, but the disadvantage of L-dopa is that this positive effect is followed by severe side effects such as dyskinesia. The search for a new drug in the treatment of PD is limited to compounds which decrease the side effects of the drugs used in the treatment of the disease, such as L-dopa-induced dyskinesia. One possible explanation for pharmaceutical companies not developing new drugs to stop disease development is because the mechanism which induces the loss of dopaminergic neurons containing neuromelanin of the nigrostriatal system is still unknown. The discovery of genes (alpha-synuclein, parkin, pink-1, DJ-1, LRRK2, GBA1, etc.) associated with familial forms of PD resulted in an enormous input into basic research in order to understand the role of these proteins in the disease. It is generally accepted that the loss of dopaminergic neurons containing neuromelanin involves mitochondrial dysfunction, protein degradation dysfunction, the aggregation of alpha-synuclein to neurotoxic oligomers, oxidative neuroinflammation and endoplasmic reticulum stress, but the question of what induces these mechanisms remains unanswered. Aminochrome, the product of dopamine oxidation and the precursor of neuromelanin, is directly involved in five of the six mechanisms and may be a better PD preclinical model.

## Palabras clave

**Palabras clave de autor:** Dopamine; drug metabolism; o-quinones; aminochrome; glutathione transferase M2-2; DT-diaphorase; Parkinson's disease; neurodegeneration

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