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Contribution of optogenetics in mitochondrial dysfunction in parkinson's disease: A new therapeutic perspective

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Parkinson's disease is a chronic, slowly progressive neurodegenerative condition. It is related to the progressive disappearance of the dopaminergic neurons of the black substance, essential to the control of the movements of the body. Although the exact cause of parkinson's disease remains unknown, evidence from neuroscientific research suggests that mutations in certain genes, including the gene for parkin and the *PINK1* gene, play an important role in the evolution of parkinson's disease. These genes help preserve the activity they exert on the mitochondria, cellular structures responsible to produce energy. Indeed, mitochondria, power plants must eliminate their protein waste damaged by oxidation. Thus, this mitochondrial process is coded by these two genes whose products are proteins called cargo because they are responsible for transporting this waste to the outside of the mitochondria. If this cleaning process is not performed properly, the mitochondrion self-ligates and the damage to the cell is considerable. And we know that when nerve cells are hungry for energy, we can easily consider the consequences. As a result, a mutation in these genes causes the degeneration of dopaminergic neurons, which predisposes to the onset of parkinson's disease. Today, we are not unaware that taking oral pharmacological treatment for parkinson's disease causes, after a few years, side effects sometimes very disabling. These adverse effects are related to intermittent intake of L-DOPA. Therefore, finding a solution to this process of neuronal death is one of the main unanswered questions in parkinson's research: Optogenetics is a technique of genetically modifying cells to make them reactive. Light, represents one of the emerging and promising strategies that has evolved over the last decade offering a new field of research to expand the therapeutic arsenal of parkinson's disease. In the native state, parkin is tightly folded back on itself, making it inactive. It cannot fulfill her role. To be active, it must undergo a conformational change. The goal of this work is to use optogenetics to activate parkin and thus prevent the death process of dopaminergic neurons in parkinson's disease.

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