



A New Therapeutic Approach to Parkinson's disease

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DESCRIPTION

Parkinson's disease (PD) is a common neurodegenerative disease. Age is the greatest risk factor, with prevalence increasing from 1% for ages 45-54 to 2-4% for ages 85 and older. Some predict that the growing population will face a "PD pandemic" that will double the prevalence over the next 20 years. Therefore, there is an urgent need for effective treatments to reduce the burden of illness. In this special issue of Neuropharmacology, invited authors review the current and new goals of pharmacological treatment of Parkinson's disease and cover the area of disease correction. Treatment of the underlying disease process up to symptomatic treatment is for the motile or non-motile symptoms of the disease. The special issue emphasizes that there are significant ongoing activities in all of these potential indications, with different targets being identified and validated to varying degrees. Parkinson's disease has been and remains an important area of study for neuro pharmacologists in both preclinical and clinical settings.

Lowering, preventing or reversing disease progression of PD remains elusive goals. Currently there are no approved interventions that have shown evidence of neuro protection in PD. To date, challenges have existed in translating preclinical advances into clinical practice. This lack of translation highlights the need to employ the most appropriate preclinical models that best mimic the relevant human neurodegenerative processes for any given target/indication; the potential value of biomarkers; to better measure target engagement and effects of interventions and the need to identify patient populations appropriate for the stage of the disease modeled by the preclinical work. Many approaches are now being investigated that will hopefully improve this pathway of development. PD heterogeneity can be challenging, from a variety of clinical features to underlying genetic causes and risk factors. That means thinking about ways to approach both the neurodegenerative process (and the symptomatic treatment of the disease) and the concept of PDPD that is longer effective as a single disease.

PD is the degeneration of midbrain dopaminergic neurons and the presence of Lewy bodies with the accumulation of proteins, including synuclein. Many potential approaches are available to counteract the effects of synuclein by reducing protein levels directly, indirectly, or through secondary effects that correct inflammation. This study outlines current and future goals for immunotherapeutic approaches that use active and passive immunization to reduce synuclein. To date, early-stage research endpoints have reported varying successes. Research is ongoing. There are no major safety concerns. A key issue for the success of immunotherapy is that endpoints such as CSF levels of synuclein and antibody levels against synuclein must reflect the burden of the disease. Other ways to promote neuroprotection, including reducing neuroinflammation; targeting lysosomal dysfunction and neurotrophic factors has been described. Various agents, such as the iron chelator and the cAbl inhibitor nilotinib, are in clinical trials and have been used in several studies so far, but the results are inconsistent. The association between diabetes and Parkinson's disease has investigated GLP1 agonists that may have multiple mechanisms of action, including improved insulin resistance. The GLP1 agonist exenatide is safe and tolerated in early clinical trials, and more trials are underway. Other GLP1 agonists are in the early stages of development. A more recent approach has been to alter the gut microbiota with the aim of preventing connections between the gut brain axis and the neurodegenerative process. Inconsistent results mean that this approach needs to be further investigated.

Other potential means for neuro protection include treatments that target known genetic risk factors associated with Parkinson's disease. All of these genetic variations are associated with multiple signaling pathways involved in PD neuro degeneration, including lysosomal and mitochondrial homeostatic disorders, vesicle transport disorders, accelerated synuclein proliferation, and inflammation. GBA variants are a common risk factor for PD and occur in 5-20% of PD.

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