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Original Article

Emerging Role Of Dulaglutide In Parkinson's Disease: Neuroprotective Mechanisms And Therapeutic Advances

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ABSTRACT

Parkinson's disease (PD) is the second most common neurodegenerative disorder and is characterized by progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta. Current pharmacological therapies mainly provide symptomatic relief and do not prevent disease progression. Recently, glucagon-like peptide-1 (GLP-1) receptor agonists have attracted considerable attention because of their neuroprotective properties. Dulaglutide, a long-acting GLP-1 receptor agonist approved for type 2 diabetes mellitus, has demonstrated antioxidant, anti-inflammatory, anti-apoptotic, and mitochondrial protective effects in experimental studies. Activation of GLP-1 receptors enhances neuronal survival through the PI3K/Akt and cAMP/PKA signaling pathways while reducing oxidative stress and neuroinflammation. These mechanisms may protect dopaminergic neurons and slow the progression of Parkinson's disease. Although clinical evidence specifically evaluating Dulaglutide in PD remains limited, findings from preclinical studies and research on other GLP-1 receptor agonists suggest promising therapeutic potential. This review summarizes the pathophysiology of Parkinson's disease, the mechanism of GLP-1 receptor agonists, the neuroprotective actions of Dulaglutide, available clinical evidence, and future research directions. Overall, Dulaglutide represents a potential disease-modifying strategy that may complement conventional dopaminergic therapy in Parkinson's disease.

Keywords: Parkinson's disease; Dulaglutide; GLP-1 receptor agonist; Neuroprotection; Dopamine; Oxidative stress; Neuroinflammation.

INTRODUCTION

Parkinson's disease (PD) is the second most common neurodegenerative disorder after Alzheimer's disease and affects millions of people worldwide. It is characterized by the progressive loss of dopaminergic neurons in the substantia nigra pars compacta, resulting in dopamine deficiency and the development of motor symptoms such as resting tremor, rigidity, bradykinesia, and postural instability [1]. In addition to motor impairment, patients frequently experience non-motor manifestations including depression, anxiety, sleep disturbances, cognitive decline, and autonomic dysfunction [2].

Current therapeutic options such as levodopa, dopamine agonists, MAO-B inhibitors, and COMT inhibitors mainly provide symptomatic relief without preventing neuronal degeneration [3]. Therefore, there is an urgent need for disease-modifying therapies capable of slowing or halting disease progression.

GLP-1 receptor agonists, originally developed for the treatment of type 2 diabetes mellitus, have shown significant neuroprotective effects in experimental models of neurodegenerative diseases. Dulaglutide is a long-acting GLP-1 receptor agonist that exhibits anti-inflammatory, antioxidant, anti-apoptotic, and mitochondrial protective properties [4]. These mechanisms suggest that Dulaglutide may preserve dopaminergic neurons and improve neurological outcomes in Parkinson's disease.

This review discusses the pathophysiology of Parkinson's disease, the mechanism of GLP-1 receptor agonists, the neuroprotective effects of Dulaglutide, current evidence from experimental and clinical studies, and its future therapeutic potential [5].

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PATHOPHYSIOLOGY OF PARKINSON'S DISEASE

Parkinson's disease (PD) is characterized by the progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta, leading to dopamine depletion in the striatum. This dopamine deficiency is responsible for the cardinal motor symptoms of the disease, including resting tremor, bradykinesia, muscular rigidity, and postural instability [6].

The pathological hallmark of Parkinson's disease is the accumulation of misfolded α-synuclein protein, which forms intracellular inclusions known as Lewy bodies. These protein aggregates impair neuronal function, disrupt synaptic transmission, and promote neuronal degeneration [7].

Oxidative stress is a major contributor to disease progression. Excessive production of reactive oxygen species (ROS) damages cellular lipids, proteins, and DNA, resulting in mitochondrial dysfunction and neuronal apoptosis [8].

Mitochondrial dysfunction, particularly impairment of Complex I of the electron transport chain, decreases ATP production and increases ROS generation, making dopaminergic neurons highly susceptible to degeneration [9].

Neuroinflammation also plays a crucial role in PD. Activated microglia release pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6, which further enhance oxidative stress and neuronal injury [10].

Excitotoxicity caused by excessive glutamate signaling leads to calcium overload, activation of apoptotic pathways, and progressive neuronal death. Genetic mutations and environmental toxins further accelerate these pathological processes [11].

Understanding these mechanisms has led to the investigation of neuroprotective therapies such as GLP-1 receptor agonists. Dulaglutide has shown potential to reduce oxidative stress, suppress neuroinflammation, improve mitochondrial function, and inhibit apoptosis, thereby protecting dopaminergic neurons in experimental models of Parkinson's disease [12].

Current Pharmacological Treatment of Parkinson's Disease

Current pharmacological treatment of Parkinson's disease (PD) primarily focuses on relieving motor and non-motor symptoms by restoring dopaminergic neurotransmission. Although these therapies significantly improve patients' quality of life, they do not stop the progressive degeneration of dopaminergic neurons. Therefore, the development of disease-modifying therapies remains a major research priority [13].

Levodopa

Levodopa remains the most effective and widely prescribed drug for the management of Parkinson's disease. After crossing the blood-brain barrier, levodopa is converted into dopamine, thereby replenishing depleted dopamine levels in the striatum. It is usually administered with a peripheral dopa-decarboxylase inhibitor such as carbidopa or benserazide to increase its bioavailability and reduce peripheral adverse effects. Long-term treatment, however, is associated with complications including motor fluctuations, wearing-off phenomenon, and levodopa-induced dyskinesia [14].

Dopamine Agonists

Dopamine agonists directly stimulate dopamine receptors and are commonly used either as monotherapy in early-stage PD or in combination with levodopa during advanced stages. Commonly prescribed agents include pramipexole, ropinirole, rotigotine, and apomorphine. These drugs reduce motor symptoms and delay the need for high-dose levodopa therapy. However, adverse effects such as nausea, orthostatic hypotension, hallucinations, excessive daytime sleepiness, and impulse-control disorders may limit their use [15].

MAO-B and COMT Inhibitors

Monoamine oxidase-B (MAO-B) inhibitors, including selegiline, rasagiline, and safinamide, prolong dopamine activity by inhibiting its metabolism in the brain. Catechol-O-methyltransferase (COMT) inhibitors such as entacapone, opicapone, and tolcapone reduce the peripheral breakdown of levodopa, thereby extending

its duration of action and reducing "off" periods. These drugs are mainly used as adjunctive therapy in patients experiencing motor fluctuations [16].

Other Therapeutic Agents

Amantadine is an NMDA receptor antagonist that is effective in reducing levodopa-induced dyskinesia and provides modest improvement in motor symptoms. Anticholinergic drugs, including trihexyphenidyl and benztropine, are mainly beneficial for tremor-predominant Parkinson's disease, particularly in younger patients. However, their use is limited because of adverse effects such as dry mouth, constipation, urinary retention, blurred vision, and cognitive impairment, especially in elderly individuals [17].

Limitations of Current Therapy

Despite substantial advances in symptomatic treatment, currently available medications do not prevent progressive neuronal degeneration or alter the natural course of Parkinson's disease. Long-term treatment is frequently associated with declining efficacy and treatment-related complications. Consequently, increasing attention has focused on novel neuroprotective agents capable of targeting neuroinflammation, oxidative stress, mitochondrial dysfunction, and α-synuclein aggregation. Among these, GLP-1 receptor agonists, particularly dulaglutide, have emerged as promising candidates because of their multiple neuroprotective mechanisms demonstrated in experimental studies [18].

GLP-1 Receptor Agonists:

Overview and Neuroprotective Mechanisms

Glucagon-like peptide-1 (GLP-1) is an incretin hormone secreted primarily by intestinal L-cells following food intake. It regulates glucose homeostasis by stimulating glucose-dependent insulin secretion, suppressing glucagon release, delaying gastric emptying, and reducing appetite. In addition to its metabolic actions, GLP-1 receptors are widely expressed in the central nervous system, including the hippocampus, cortex, hypothalamus, and substantia nigra. Activation of these receptors has been shown to promote neuronal survival and improve brain function, making GLP-1 receptor agonists promising therapeutic agents for neurodegenerative disorders such as Parkinson's disease [19].

GLP-1 receptor agonists (GLP-1RAs) are synthetic analogues of endogenous GLP-1 that are resistant to degradation by dipeptidyl peptidase-4 (DPP-4). Commonly available GLP-1RAs include exenatide, liraglutide, dulaglutide, semaglutide, and lixisenatide. Initially developed for the treatment of type 2 diabetes mellitus, these drugs have demonstrated significant neuroprotective effects in experimental studies through multiple molecular mechanisms [20].

Anti-inflammatory Effects

Neuroinflammation is a key pathological feature of Parkinson's disease. Activated microglia release inflammatory cytokines such as TNF-α, IL-1β, and IL-6, which contribute to neuronal injury and disease progression. GLP-1 receptor agonists suppress microglial activation and inhibit inflammatory signaling pathways, including NF-κB, thereby reducing neuroinflammation and protecting dopaminergic neurons from further damage [21].

Reduction of Oxidative Stress

Oxidative stress contributes significantly to dopaminergic neuronal degeneration in Parkinson's disease. Excessive production of reactive oxygen species (ROS) damages lipids, proteins, and DNA, ultimately leading to neuronal death. GLP-1 receptor agonists enhance endogenous antioxidant defenses by increasing the activities of superoxide dismutase (SOD), catalase, and glutathione peroxidase, while simultaneously reducing ROS production. These antioxidant actions help preserve neuronal integrity [22].

Improvement of Mitochondrial Function

Mitochondrial dysfunction is closely associated with the pathogenesis of Parkinson's disease. GLP-1 receptor activation improves mitochondrial biogenesis, enhances ATP production, stabilizes mitochondrial membrane potential, and reduces mitochondrial oxidative damage. Improved mitochondrial function enhances neuronal survival and delays disease progression [23].

Anti-apoptotic Activity

Programmed neuronal cell death plays a major role in Parkinson's disease progression. GLP-1 receptor agonists activate intracellular survival pathways such as PI3K/Akt and cAMP/PKA, resulting in increased expression of anti-apoptotic proteins (Bcl-2) and decreased expression of pro-apoptotic proteins (Bax and caspase-3). These effects reduce neuronal apoptosis and improve cell survival [24].

Promotion of Neurogenesis and Synaptic Plasticity

Experimental studies indicate that GLP-1 receptor agonists promote neuronal differentiation, synaptic plasticity, and neurogenesis. These effects may improve learning, memory, and motor function while supporting neuronal repair following neurodegeneration. Enhanced synaptic function may also contribute to improved neurological outcomes in Parkinson's disease [25].

Reduction of α-Synuclein Aggregation

Accumulation of misfolded α-synuclein proteins and Lewy body formation are characteristic pathological features of Parkinson's disease. GLP-1 receptor agonists promote autophagy and lysosomal degradation pathways, facilitating the clearance of abnormal protein aggregates. This mechanism may reduce neuronal toxicity and slow disease progression [26].

Therapeutic Significance

Because GLP-1 receptor agonists simultaneously target neuroinflammation, oxidative stress, mitochondrial dysfunction, apoptosis, and protein aggregation, they represent an attractive disease-modifying strategy for Parkinson's disease. Among this drug class, dulaglutide has received increasing attention due to its long half-life, convenient once-weekly administration, and favorable safety profile. These characteristics support its further investigation as a potential neuroprotective therapy in Parkinson's disease [27].

Dulaglutide: Pharmacology and Mechanism of Neuroprotection

Dulaglutide is a long-acting glucagon-like peptide-1 receptor agonist (GLP-1RA) approved for the treatment of type 2 diabetes mellitus. It is composed of two modified GLP-1 analogue chains linked to a human immunoglobulin G4 (IgG4) Fc fragment, which protects the molecule from degradation by dipeptidyl peptidase-4 (DPP-4). This structural modification prolongs its plasma half-life and permits once-weekly subcutaneous administration. In addition to its glucose-lowering effect, Dulaglutide has shown promising neuroprotective activity in experimental models of Parkinson's disease [28].

Following GLP-1 receptor activation, Dulaglutide stimulates intracellular signalling pathways including PI3K/Akt, cAMP/PKA, and MAPK/ERK. These pathways enhance neuronal survival, improve mitochondrial function, promote neurotrophic factor expression, and suppress apoptotic cell death. Consequently, dopaminergic neurons become more resistant to neurodegenerative injury [29].

Dulaglutide also reduces oxidative stress by limiting reactive oxygen species generation and strengthening endogenous antioxidant defence systems. Simultaneously, it suppresses microglial activation and decreases the release of inflammatory mediators such as TNF-α, IL-1β, and IL-6, thereby reducing chronic neuroinflammation associated with Parkinson's disease [30].

Recent preclinical investigations further suggest that Dulaglutide enhances autophagy, improves clearance of abnormal α-synuclein aggregates, and restores mitochondrial bioenergetics. These combined actions may slow the degeneration of dopaminergic neurons and delay disease progression. Nevertheless, large-scale randomized clinical trials are still required to establish its long-term efficacy and safety in patients with Parkinson's disease [31].

Preclinical and Clinical Evidence

Several preclinical studies have demonstrated that GLP-1 receptor agonists exert neuroprotective effects in experimental models of Parkinson's disease. In toxin-induced animal models, these agents preserved dopaminergic neurons, improved motor performance, reduced oxidative stress, and attenuated neuroinflammatory responses. The findings indicate that activation of GLP-1 receptors may delay neuronal degeneration and improve functional outcomes [32].

Among the available GLP-1 receptor agonists, Dulaglutide has shown encouraging results in laboratory investigations. Experimental evidence suggests that Dulaglutide improves mitochondrial function, suppresses apoptotic signalling, and enhances neuronal survival by activating intracellular pathways involved in cell protection. These effects may help maintain dopaminergic neuronal integrity during disease progression [33].

Clinical studies evaluating GLP-1 receptor agonists, particularly Exenatide and Lixisenatide, have reported improvements in motor symptoms and slower clinical deterioration in some patients with Parkinson's disease. Although direct clinical evidence for Dulaglutide remains limited, its pharmacological profile and neuroprotective mechanisms suggest that it may provide similar therapeutic benefits [34].

In addition to improving motor function, GLP-1 receptor agonists may positively influence non-motor manifestations of Parkinson's disease, including cognitive impairment and neuroinflammation. Their ability to target multiple pathological mechanisms simultaneously has generated considerable interest as a potential disease-modifying approach rather than merely symptomatic treatment [35].

Despite these promising observations, the current clinical evidence is still insufficient to establish Dulaglutide as a standard therapy for Parkinson's disease. Large, multicentre, randomized controlled trials with extended follow-up are required to determine its long-term efficacy, optimal dosage, and safety profile before routine clinical use can be recommended [36].

Advantages and Limitations of Dulaglutide in Parkinson's Disease

Dulaglutide offers several pharmacological advantages that make it an attractive candidate for neuroprotection in Parkinson's disease. Its once-weekly administration improves patient adherence compared with medications requiring daily dosing. The drug also exhibits a prolonged plasma half-life, sustained GLP-1 receptor activation, and a well-established safety profile in patients with type 2 diabetes mellitus. Experimental findings suggest that Dulaglutide simultaneously targets multiple pathological mechanisms, including oxidative stress, mitochondrial dysfunction, neuroinflammation, and apoptosis, which are central to the progression of Parkinson's disease [37].

Another important advantage is its ability to enhance neuronal survival by activating intracellular signalling pathways such as PI3K/Akt and cAMP/PKA. These pathways promote cellular repair, improve mitochondrial function, and reduce inflammatory mediator production. Unlike conventional dopaminergic therapies that primarily relieve symptoms, Dulaglutide has the potential to modify the underlying disease process by protecting dopaminergic neurons from progressive degeneration [38].

Despite these promising characteristics, several limitations remain. Clinical evidence specifically evaluating Dulaglutide in Parkinson's disease is still limited, and most available data are derived from preclinical studies or investigations involving other GLP-1 receptor agonists. Furthermore, the optimal treatment duration, appropriate dosage, long-term neurological benefits, and safety in non-diabetic patients require further evaluation through large-scale randomized controlled trials. These limitations must be addressed before Dulaglutide can be routinely recommended as a disease-modifying therapy for Parkinson's disease [39].

FUTURE PERSPECTIVES

The growing evidence supporting the neuroprotective potential of GLP-1 receptor agonists has opened new avenues for the treatment of Parkinson's disease. Future research should focus on conducting large-scale, multicentre, randomized controlled clinical trials to evaluate the long-term efficacy and safety of Dulaglutide in patients with Parkinson's disease. Such studies are essential to determine whether Dulaglutide can slow disease progression rather than merely provide symptomatic improvement [40].

Further investigations are also required to identify the optimal dosage, treatment duration, and appropriate stage of disease at which Dulaglutide may provide maximum therapeutic benefit. The discovery of reliable biomarkers for monitoring neuronal protection and treatment response could improve patient selection and facilitate personalized therapeutic strategies [41].

In addition, future studies should explore the potential of combination therapy, where Dulaglutide is administered alongside conventional antiparkinsonian medications such as levodopa or dopamine agonists. This approach may enhance therapeutic outcomes by simultaneously improving symptomatic control and reducing progressive neuronal degeneration. Advances in molecular biology and precision medicine are expected to further clarify the role of GLP-1 receptor agonists as disease-modifying agents in Parkinson's disease [42].

CONCLUSION

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by the selective degeneration of dopaminergic neurons in the substantia nigra, leading to both motor and non-motor impairments. Although currently available therapies effectively improve symptoms, they do not prevent the progressive loss of neurons or alter the underlying disease process. Therefore, the development of disease-modifying therapies remains a major objective in Parkinson's disease research [43].

Dulaglutide, a long-acting glucagon-like peptide-1 receptor agonist (GLP-1RA), has emerged as a promising therapeutic candidate because of its multiple neuroprotective mechanisms. Experimental studies have demonstrated that dulaglutide reduces neuroinflammation, oxidative stress, mitochondrial dysfunction, and neuronal apoptosis while promoting neuronal survival and improving motor function. These findings suggest that dulaglutide may provide benefits beyond conventional symptomatic treatment by targeting the underlying pathological mechanisms of Parkinson's disease [44].

Although preclinical evidence is highly encouraging, clinical studies specifically evaluating dulaglutide in Parkinson's disease remain limited. Large, multicenter, randomized controlled clinical trials are required to establish its long-term efficacy, safety, optimal dosage, and disease-modifying potential. Future research should also focus on identifying reliable biomarkers and evaluating combination therapies to maximize therapeutic outcomes [45].

In conclusion, dulaglutide represents a promising and innovative therapeutic strategy for Parkinson's disease. Its multitarget neuroprotective actions provide a strong scientific rationale for continued investigation. If future clinical trials confirm its efficacy, dulaglutide may become an important disease-modifying therapy capable of slowing disease progression and improving the quality of life of patients with Parkinson's disease [46].

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Naveen Kumar: Literature review, validation of scientific content, and manuscript review.

Indumati Thakare: Supervision, methodology, critical review, and final approval of the manuscript.

Madhuri Baghel: Data curation, reference management, manuscript editing, and formatting.

Hari Prasad Sonwani: Conceptualization, supervision, critical revision, project administration, and final approval of the manuscript.

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