INTI JOURNAL | Vol.2026, Issue 2, No.5
eISSN:2600-7320

A Review on Exploring the Role of *Solanum Nigrum L* in the Management of Parkinson Disease

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Abstract

Solanum nigrum L. (black nightshade) has gained significant attention for its potential to help with Parkinson's disease (PD). This disease is marked by the selective loss of dopaminergic neurons in the substantia nigra. The causes of PD involve oxidative stress, ongoing neuroinflammation, problems with mitochondria, and abnormal protein buildup. Traditional treatments like levodopa provide only temporary relief and come with serious side effects. Because of this, researchers are looking into multi-targeted plant-based options. S. nigrum has a diverse chemical profile that helps counteract these neurodegenerative processes. Its steroidal glycoalkaloids, such as solanine and solamargine, reduce neuroinflammation by inhibiting NF-κB signaling. Its flavonoids, like quercetin and kaempferol, along with phenolic acids, lessen reactive oxygen species (ROS) and lipid damage while also stabilizing dopamine levels. Moreover, its lignanamides can impact autophagy, possibly reducing α-synuclein buildup. In addition, its saponins, carotenoids, and polysaccharides provide further immune support and prevent cell death. This review brings together the ethnopharmacological, phytochemical, and experimental insights about S. nigrum. It emphasizes the need for more detailed studies and clinical tests to confirm its potential as a complementary treatment for PD.

Keywords

Solanum nigrum L., Parkinson's disease, Neuroprotection, Oxidative stress, Traditional medicine

Introduction

The second most common neurodegenerative disease in the world is the Parkinson disease which has millions of fans and has significant healthcare and family burdens. The disease is a complicated combination of motor and non-motor symptoms that seriously deteriorate with time and seriously interrupt the quality of life of people who have the illness. It is important to understand the

Submission: 10 May 2026; **Acceptance:** 16 July 2026; **Available online:** July 2026

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underlying mechanics of this debilitating condition in order to come up with effective therapeutic interventions.

The hallmark pathophysiology of the Parkinson disease implies gradual destruction of dopaminergic cells in the substantia nigra pars compacta, which is one of the most significant areas of the midbrain that produces dopamine. Research has shown that about 50 to 80 percent of the dopaminergic neurons in this part have already degenerated by the time clinical motor symptoms appear. Morris et al. (2024). This massive cell death causes a tremendous loss of striatal dopamine levels, which, in turn, interferes with the complex neural networks governing movement in the basal ganglia. The resultant dopamine deficit presents itself as the cardinal motor signs of the disease such as bradykinesia, resting tremor, rigidity, and postural instability.

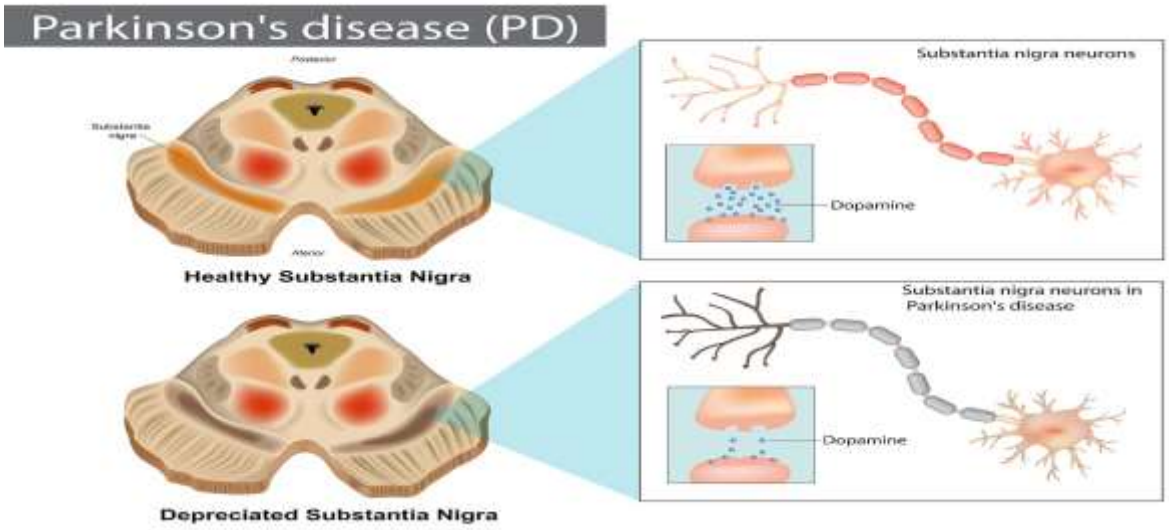

Figure 1: Illustration of dopaminergic neuron degeneration in Parkinson disease, showing healthy substantial nigra neurons with normal dopamine release (top) versus depleted substantial nigra neurons with reduced dopamine in Parkinson disease (bottom)

In addition to the established loss of dopaminergic neurons, the neuropath logical pathogenesis of Parkinson disease also includes the abnormal accumulation and aggregation of alpha-syncline protein, which is the typical structure of Lowy bodies and Lowy neuritis found in the injured areas of the brain (Figure 1). These inclusion bodies are proteinaceous bodies which are also a characteristic pathological feature of the disease. The aggregation of alpha-syncline seems to have a prion-like spread, with the onset at the lower part of the brainstem and olfactory bulb and then progression to the midbrain and finally to the neocortex. Dong-Chen et al. (2023). This gradual development of the pathology is associated with advancement of symptoms which in the more severe forms are characterized by the more complicated processes of thinking and autonomous activity.

Molecular processes, which lead to neurodegeneration in Parkinson disease, are much more than dopamine loss and protein aggregation. It has also been shown that there are various interrelated pathogenic pathways, such as mitochondrial dysfunction, disrupted autophagy-

lysosomal degradation, endoplasmic reticulum stress, and oxidative damage. (Zafar & Yaddanapudi; Yaddanapudi et al.(2023).

Neuroinflammation has become a major cause of disease development. There are both natural and adaptive immune responses involved in the pathogenic cascade. Microglia, the innate immune cells of the brain, are activated and increase the expression of pro-inflammatory cytokines such as interleukin-1 bacterial and tumor necrosis factor-alpha by increasing inflammatory signaling pathways, which include nuclear factor kappa-B and NLRP3 inflammasome. Dong-Chen et al. (2023).

These proinflammatory agents promote a death microenvironment that enhances the death of dopaminergic neurons. Also, CD4 and CD8 T cell lymphocyte infiltration in the substantial nigra has been identified in patients with Parkinson disease, and in the early phases of the disease, CD8 T cells are found to be of high concentration. Morris et al. (2024).

The environmental factors have been cited as the significant causative factors of Parkinson disease. The epidemiological researchers have developed a strong correlation between exposure to pesticides and herbicides and the risk of diseases. In particular, parquet, a herbicide, has been widely examined in animal and cellular models, making the herb cause parkinsonian symptoms via oxidative stress schemes and dopaminergic neuron selective toxicity. Maitra et al. (2021). On the same note, the pesticide rotenone suppresses the mitochondrial complex I, inducing the formation of the reactive oxygen species and dopaminergic cells death. Such environmental toxins can contribute with some genetic susceptibility factors generating a multifactorial disease process that rationalizes the heterogeneity in the presentation and progression of the disease.

Other Compounds and Plants used as Drugs in Parkinson Disease.

Conventional dopaminergic therapy and the multifactoriality of the Parkinson disease have led to widespread research on plant-based therapeutic options. Medicinal Natural products based on natural plants have their own benefits, such as multi-target actions of the drug, relatively good safety profiles and possible disease-modifying effects other than simple symptomatic action. The use of a number of botanical remedies to treat the neurological disorders has long been used in traditional medicine systems of various cultures, and contemporary scientific studies have been starting to verify many of these traditional uses as well as explaining their molecular pathways (Table 1).

Table 1: Major medicinal plants and their bioactive compounds investigated for Parkinson disease therapy

Plant Species	**Active Compounds**	**Mechanism of Action**	**Key References**
Mucuna pruriens	L-DOPA, alkaloids	Dopamine replacement, antioxidant	Khazdair et al.(2020)
Ginkgo biloba	Flavonoids, ginkgolides	Antioxidant, anti-inflammatory	Corona et al.(2018)
Bacopa monnieri	Bacoside A, bacosides	Antioxidant, neuroprotective	Srivastav et al. (2017)
Curcuma longa	Curcumin, turmerone	Anti-inflammatory, MAO inhibition	Khazdair et al. (2020)

Withania somnifera	Withanolides	Neuroprotective, antioxidant	Srivastav et al., (2017)
Camellia sinensis	Catechins, EGCG	Antioxidant, anti-aggregation	Yang et al. (2024)
Panax ginseng	Ginsenosides	Neuroprotective, anti-apoptotic	Javed et al. (2019)
Centella asiatica	Asiaticoside, madecassoside	Anti-aggregation, antioxidant	Javed et al.(2019)

Some flavonoid extracts that were obtained in different plant materials have demonstrated potential in preclinical research of Parkinson disease (Figure2).A more common antioxidant, quercetin, raised striatal dopamine and lowered dopaminergic neuronal losses in 6-hydroxydopamine rat models with an increase in antioxidant enzyme activities. Corona et al., (2018). Kaempferol, a plant compound present in grapefruit and other sources, enhanced motor coordination, increased striatal dopamine and metabolite levels and inhibited the loss of neurons in the MPTP-induced models labeled with tyrosine hydroxylase. A flavones, baicalein, of Scutellaria species, was able to stop the abnormal behavior because it increased dopaminergic neurons and dopamine levels in striatum and inhibited oxidative stress and astroglial responses.

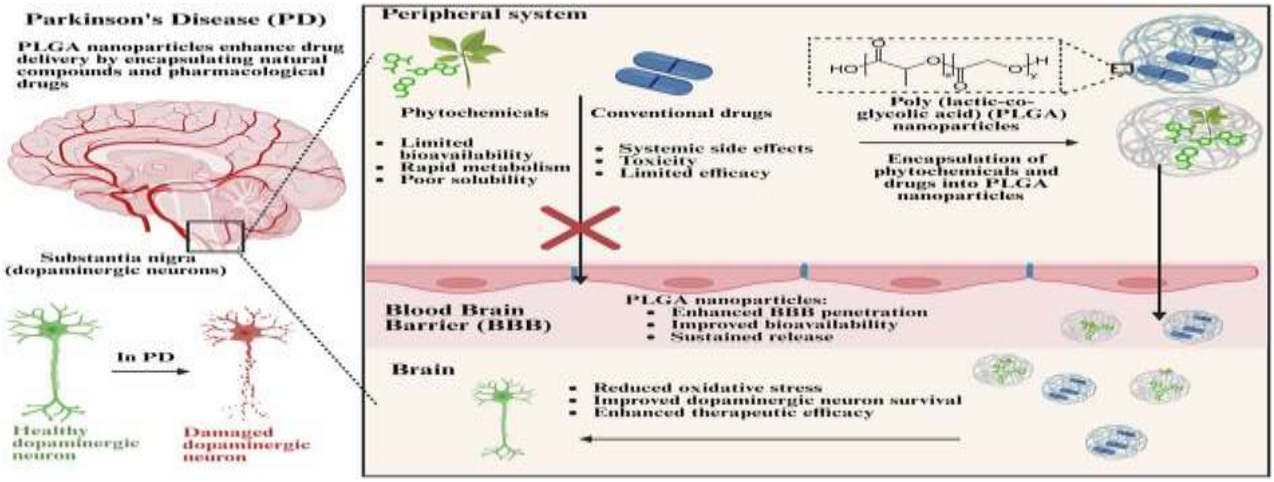

Figure2: Role of PLGA nanoparticles in improving therapeutic delivery to the brain for Parkinson's disease management

Alpha-synuclein aggregation natural products are a relatively promising type of therapeutic approach.Several plant extracts and single compounds have also been shown to prevent alpha-synuclein oligonucleation, fibrillation, and aggregation and promote disaggregation of fibrils already formed.It has been demonstrated that cinnamon extract can prevent the aggregation of alpha-synuclein, stabilize soluble oligomers, and force aggregation to an off-pathway oligomer that is less toxic. Javed et al. (2019).Cinnamon treatment of Drosophila models expressing A53T mutant alpha-syncline enhanced behavior and cognition and decreased pathological aggregates. On the same note, the extracts of Centella asiatica prevented alpha-syncline aggregation, oligomers stabilization, and preformed fibrils disintegration in the biochemical experiments.

Among the new neuroprotectants against the environmental toxin induced Parkinson disease is the polymethoxyflavonoid gardenin A derived out of the Gardenia resinifera. The research conducted on paraquat induced Drosophila models showed that gardenin A was highly effective in enhancing survival, decreasing mobility defects, and inhibiting dopaminergic neuron death. Maitra et al. (2021).More to the point,the neuroprotective effect is not confined to mere antioxidant potential, but it entails interference with neuroinflammatory events, namely,the NF-kappa cascade and the synthesis of nitric oxide. This observation demonstrates the inaccuracy of the classical role of plant polyphenols as direct radical scavengers and the neurohormesis hypothesis, according to which phytochemicals induce adaptive stress response pathways at the right concentrations.

Potential of *Solanum nigrum L.* Plant against Parkinson Disease.

Solanum nigrum L. also referred to as American black nightshade or the glossy nightshade, is a member of the *Solanaceae* family, which contains more than 2000 species spread worldwide in tropical and subtropical areas. It is a herbaceous plant that has been widely used in traditional medicine systems in the treatment of a variety of diseases such as inflammatory diseases, infections, and neurological diseases. Recent scientific studies have commenced on explaining the neuroprotective effects of *Solanum nigrum L.* and other closely related *Solanum* species and are showing promising prospects of treatment of Parkinson disease due to their abundant phytochemical constituents and various mechanism of action.

Solanum nigrum L. exhibits the typical diversity of the *Solanaceae* family: the phytochemical composition of the plant includes large amounts of steroidal alkaloids, glycoalkaloids, flavonoids, phenolic compounds and saponins. The flavonoids, alkaloids, tannins, saponins, and phytosterols have been validated in methanol extracts of leaves of *Solanum nigrum L.* L. Ukwubile et al.(2024).Quantitative analysis indicated phenolics content of 345.12 milligrams of gallic acid equivalent per gram and flavonoid content of 88.24 milligrams of quercetin equivalent per gram. These concentrations of polyphenolic compounds play a key role in the antioxidant activity of the plant, a factor that is important as an anti-oxidant agent in neuroprotecting neurons against oxidative stress-induced neuronal damage that is typical of Parkinson disease. The most pharmacologically important group of *Solanum* species compounds is the steroidal alkaloid group of compounds. To date, it has been found that the structure of approximately 90 different alkaloids has been recorded in 350 *Solanum* species. Manoharan et al.(2024). *Solanum nigrum L.* and the closely related species produce the primary glycoalkaloid compounds namely solamargine, solasonine, solanine, and solanidine. These compounds have a steroidal structure with nitrogen-based moieties, which give them the characteristic of an alkaloid and a steroid. It has been proved that *Solanum* alkaloids have various biological properties, such as anticancer, anti-inflammatory, antimicrobial, analgesic, and most importantly neuroprotective. This structural diversity of these alkaloids offers the prospects of finding compounds with particular pattern of activity that can be used to target various aspects of Parkinson disease pathophysiology(Figure 3).

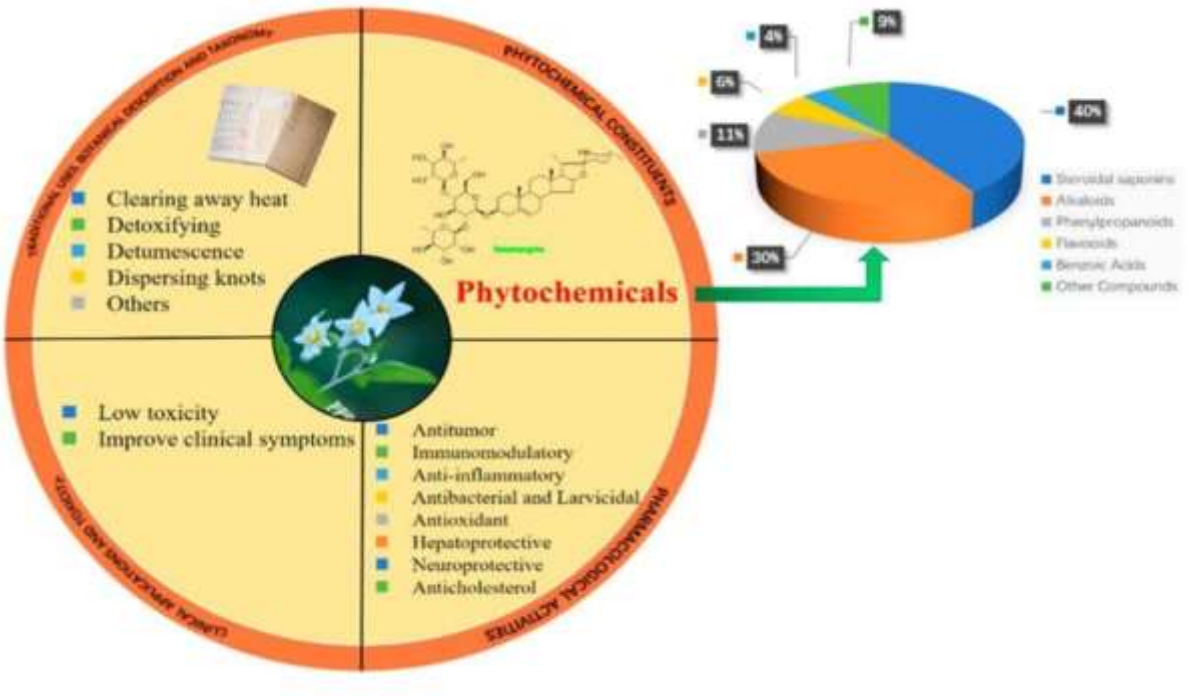

Figure 3: Mechanism of plant-derived phytochemicals in neuroprotection for Parkinson disease, illustrating enhanced blood-brain barrier penetration, improved bioavailability, reduced oxidative stress, and improved dopaminergic neuron survival through encapsulated delivery sy

The neuroprotective effect of *Solanum nigrum L.* and the other species has been evidenced using several experimental methods. Investigations on *Solanum nigrum L.* which is a related species with a similar phytochemical composition demonstrated considerable neuroprotective effects against diverse neurotoxic challenges. The effect of *Solanum nigrum L.* extract on the ATP-induced cytotoxicity in PC12 neuronal cells showed that pretreatment after 72 hours had significant neuroprotective effects because it inhibited apoptosis and stimulated cell proliferation. The extract inhibited ATP-induced neuronal cell death by the means of calcium signaling regulation and inhibition of the apoptotic pathway. Besides, the research carried out on rotenone-induced Parkinson disease rat models showed that *Solanum nigrum L.* treatment improved motor deficits and offered dopaminergic neuroprotection. Iftikar et al.(2024).

One finding is especially applicable, and it was based on the study of *Solanum* vegetable-based diets, where the authors analyzed the impact of African egg plant and black nightshade leafy vegetables on models of neurodegenerative diseases. Ogunsuyi et al.(2018). The vegetables were suggested as the potential source of functional foods and nutraceuticals which could be used to prevent and manage the disease related to cognitive impairment such as Alzheimer disease and Parkinson disease. Notably, the scientific studies have received traditional support with folklore medicine which reportedly has been used in the management of Parkinson disease and loss of memory with the use of juice extracted by *Solanum nigrum L.* leaves.

Solanum nigrum L. extracts antioxidant activity is a main mechanism of neuroprotection in Parkinson disease. Radical scavenging ability and reducing power of both aqueous and alkaloid extracts of *Solanum* species had a dose-dependency in biochemical tests. Ogunsuyi et al.(2020). The aqueous extracts tended to exhibit greater antioxidant activity than the alkaloid extracts, presumably as a result of the greater concentrations of water-soluble phenolic compounds and flavonoids. The antioxidant action is especially applicable in the context of Parkinson disease where oxidative stress is the key factor in the apoptosis of dopaminergic neurons. The production of reactive oxygen species in the metabolic processes of dopamine and mitochondrial dysfunction, as well as the inflammatory process, causes a toxic environment that outgrows the endogenous antioxidant defenses. These defenses can be supplemented by plant-based antioxidants of *Solanum* species, which have the ability to scavenge free radicals, limit lipid peroxidation, and stabilize the redox situation within cells.

In addition to direct antioxidant activity, *Solanum* extracts exhibit much inhibitory activity on the enzymes monoamine oxidase and acetyl cholinesterase, which are both relevant therapeutic targets in neurodegenerative conditions. The research that examined *Solanum* species leaf extracts revealed that the aqueous and alkaloid extracts were dose-dependent inhibitors of monoamine oxidase activity. Ogunsuyi et al.(2020).The magnitude of MAO inhibition was greater in black nightshade than in African eggplant, and vice versa with the acetyl cholinesterase inhibition. Particularly,the inhibition of monoamine oxidase is applicable to treatment of Parkinson disease since the enzyme oxidizes dopamine and their inhibitors maintain the levels of dopamine in the striatum. The selective MAO-B inhibitors are now being used as an adjunctive agent in the treatment of Parkinson disease to extend the effects of levodopa and native dopamine. The fact that *Solanum* extracts have MAO inhibitor capability indicates that it may be used as not only symptomatic based on dopamine preservation but also neuroprotective based on the reduction of oxidative stress produced during dopamine metabolism.

The neuroprotective effect of *Solanum nigrum L.*on Parkinson disease also has an anti-inflammatory effect. A study that investigated the anti-inflammatory effects of *Solanum nigrum L.* methanol leaf extract revealed the effectiveness of the extract in the production of pro-inflammatory cytokines. Ukwubile et al.(2024). The inflammatory condition is essential in the development of the Parkinson disease and the involvement of the activated microglia and high levels of inflammatory mediators in the formation of neurotoxic environment within the brain accelerates the process of dopaminergic neuron death. The plant compounds which regulate the neuroinflammation can slow down the progression of the disease by decreasing the inflammatory load. The involvement of inhibition of NF-kappaB signaling, down-regulation of expression of inflammatory enzymes such as cyclooxygenase and inducible nitric oxide synthase, and inhibition of release of inflammatory cytokines are likely the anti-inflammatory effects.

Alpha-synuclein aggregation has been addressed by *Solanum* alkaloids which show particular effects on this key pathological process in Parkinson disease. Though the current research on alpha-synuclein aggregation has mostly involved other plant species, the alkaloid chemical similarity among Solanum species implies that it can be applied in other species. The chemical components extracted out of *Solanum nigrum L.* and subjected to neuroprotective effect against the hydrogen peroxide-mediated cytotoxicity in neuroblastoma cells found that the particular compounds showed notable protective results. Bai et al., (2022)(Table 2).

The pathways are probably to prevent the misfolding of alpha-synuclein, restrain the process of oligomerization and fibrillization, and possibly enhance the process of clearing of aggregates by increasing autophagy. Such effects are additional to the actions of the antioxidant and anti-inflammatory, and they offer multi-level intervention of the pathogenic cascade.

Table 2: Summary of experimental studies demonstrating neuroprotective effects of *Solanum nigrum L.* and related species in Parkinson disease-relevant models

Study Model	Extract/Compound	Observed Effects	References
PC12 cells	*S. nigrum* extract	Reduced ATP-induced cytotoxicity, inhibited apoptosis	Iftikar et al.(2024)
Rotenone rat model	*S. nigrum* extract	Improved motor function, dopaminergic protection	Iftikar et al.(2024)
Scopolamine model	*S. nigrum* diet	Reversed cognitive impairment, restored antioxidants	Ogunsuyi et al.(2018)
In vitro assays	*S. nigrum* extract	MAO and AChE inhibition, antioxidant activity	Ogunsuyi et al.(2020)
Neuroblastoma cells	*S.nigrum* compounds	Neuroprotection against H2O2 damage	Bai et al. (2022)
Cell culture	*S. nigrum* extract	Anti-inflammatory, reduced cytokine production	Ukwubile et al.(2024)

Solanum biophytochemicals have a bioavailability and pharmacokinetic profile that is of significance in therapeutic development. Most of alkaloids and flavonoids have low oral bioavailability because of low aqueous solubility, high first-pass metabolism and low intestinal absorption. Nevertheless, aqueous decoctions or food preparations that can be used in conventional methods of preparation, can increase bioavailability due to matrix effects and the inclusion of absorption enhancers. Contemporary drug delivery solutions, such as nanoparticle encapsulation, liposomal suspensions, and phytosome technologies, provide the possibilities of enhancing the delivery of Solanum bioactive substances to the central nervous system. The studies about the encapsulation of plant-derived compounds with PLGA nanoparticles have shown an increase in blood-brain barrier penetration, bioavailability, sustained delivery, and reduced side effects.

Solanum nigrum L. has a safety profile that should be carefully taken into account because the *Solanaceae* family is characterized by toxic glycoalkaloids expressed by solanine under toxic dosages or in the wrong parts of the plant. Nevertheless, the leaves and young growth of *Solanum nigrum L.* have been used as vegetables in a variety of cultures with little or no toxicity when cooked. Research has indicated that neuroprotective compounds can be obtained selectively by using controlled forms of extraction with minimum amounts of toxic component. The therapeutic index between effective and toxic doses must also be determined in terms of systematic dose-response and toxicological analyses. The use over centuries is some assurance of safety at normal levels of consumption, yet standardization of extracts and identification of safe dose ranges is necessary to the clinical development.

The similarities and distinct benefits to *Solanum nigrum L.* compounds can be compared with the established methods of treating Parkinson disease. In contrast to synthetic levodopa, which only replaces dopamine symptoms without altering disease progression, *Solanum*

phytochemicals have multi-target disease-modifying properties that include antioxidant, anti-inflammatory, anti-aggregation and neuroprotective activities. Although *Mucuna pruriens* offers natural levodopa, Solanum species vindicate compensatory responses that can potentially be useful as addictive therapy or preventations. This is an attractive therapeutic profile when combining MAO inhibition, antioxidant and anti-inflammatory action in one source of plants and is an attractive therapy when the action is aimed at several pathogenic processes at the same time.The areas of study of *Solanum nigrum L.* in Parkinson disease that need to be addressed in the future include a number of critical areas. Primarily, bioactivity-guided fractionation of systematic identification and characterization of the strongest neuroprotective compounds of *Solanum nigrum L.* extracts will allow preparing standardized preparations with homogenous activities. Second, comprehensive mechanistic research explaining the molecular targets and the signaling pathways influenced by *Solanum* compounds will be used to inform rational therapeutic development. Third, pharmacokinetic and biodistribution research is required so as to know how these compounds enter the brain and the best dosing mechanisms. Fourth, efficacy and safety experiments in animal models of Parkinson disease over a long period will prove evidence of concept of disease-modifying effects. Lastly, it will necessitate the design of appropriate clinical trials in human Parkinson disease subjects to be able to apply the preclinical research to clinical therapeutic models.

The recent accumulation of evidence in favor of the neuroprotective effects of *Solanum nigrum L.* can be viewed in the context of the general trends in the endogenous disease studies that emphasize the use of natural products as therapeutic agents and multi-target disease-modifying strategies. The synthetic drugs that can be used to treat specific pathways at a time may not be as effective as plant-based products with the ability to retain the opportunity to regulate many of the pathogenic pathways at once despite the ongoing evolution of the knowledge of the multifaceted and complicated pathogenesis. As an experimental model with its rich phytochemical profile, previous application in treatment of the neurological diseases, and previous neuroprotective properties, *Solanum nigrum L.* will be a good candidate in the future status of a complementary or alternative treatment of Parkinson disease. The example of combining traditional knowledge with the modern scientific research exemplifies that ethnopharmacological studies can help in finding new therapeutic measures to treat difficult neurodegenerative diseases.

Conclusion

The growing burden of Parkinson's disease necessitates a shift from single-target symptomatic therapies to multi-targeted, disease-modifying alternatives due to the limitations and side effects of conventional dopamine replacement drugs. This review highlights the multi-targeted neuroprotective potential of *Solanum nigrum* L., which leverages its rich phytochemical profile—including flavonoids, phenolic acids, and steroidal glycoalkaloids—to simultaneously counter oxidative stress, neuroinflammation, monoamine oxidase activity, and abnormal protein aggregation. While these preclinical findings validate its traditional use, challenges such as low oral bioavailability and potential glycoalkaloid toxicity must be addressed through controlled extraction techniques and advanced delivery systems like PLGA nanoparticles. Ultimately, *Solanum nigrum* L. represents a highly promising complementary therapeutic strategy, though systematic bioactivity-guided fractionation, long-term in vivo safety assessments, and structured

human clinical trials remain essential to fully transition this botanical asset into modern parkinsonian pharmacotherapy.

Acknowledgements

There is no grant or funding bodies to be acknowledged for preparing this paper.

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