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Original Article

Medicinal Plants in A Parkinson Disease Management

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ABSTRACT
Parkinson's disease (PD) is a common movement disorder seen in neurological practice, but diagnosis management is challenging and difficult. This PD is the is the second most common disorder and affects 2–3% of the population >60 years of age. It is more common in men than women, and uric acid also seems to be associated with lower PD. Neuronal loss in the substantia nigra, which causes striatal dopamine deficiency. It happens when nerve cells in the brain don't produce enough of a brain chemical called dopamine. Sometimes PD is caused by rare familial genetic mutations, but in most cases, it results from multiple genetic and environmental risk factors. considering the large number of motor and non-motor symptoms in PD patients. When dementia occurs, the decline is usually rapid and stereotyped. The combination of Lewy and Alzheimer disease pathophysiology is the most robust pathophysiology correlated with PDD. Currently, levodopa is the mainstay of treatment. However, long-term use of levodopa is interrupted by several side effects, like dyskinesia and on-off fluctuation. there is no cure for PD. Deep brain stimulation is one such evolving technology with proven efficacy in advanced PD and a promising result in early PD. Parkinson disease has a patient perspective on the after-disease symptoms impacting psychosocial quality of life. This review aims to present the aspects in which greater evidence exists and summarize the epidemiology, pathogenesis, clinical features, diagnosis, approach, and treatment in PD.
Keywords: Parkinson's disease, Pathophysiology, Etiology, Symptoms.

INTRODUCTION
Parkinson's disease (PD) is a progressive neurodegenerative disorder primarily affecting motor function. First described by James Parkinson in 1817 as the "Shaking Palsy," it is now recognized as the second most common neurodegenerative disease after Alzheimer's disease [1]. PD affects millions of people globally, imposing a substantial clinical, social, and economic burden. The primary motor symptoms include bradykinesia, rigidity, resting tremors, and postural instability. However, non-motor symptoms such as cognitive decline, mood disorders, sleep disturbances, and autonomic dysfunction also play a significant role in the disease's progression and impact on quality of life [2].The pathogenesis of PD is multifactorial, involving a combination of genetic predispositions and environmental factors. The hallmark pathological features of PD are the progressive loss of dopaminergic neurons in the substantia nigra pars compacta and the presence of Lewy bodies intracellular inclusions composed mainly of misfolded α-synuclein protein. These changes lead to a significant reduction in striatal dopamine levels, disrupting the normal functioning of the basal ganglia, a group of nuclei critical for motor control [3-5].

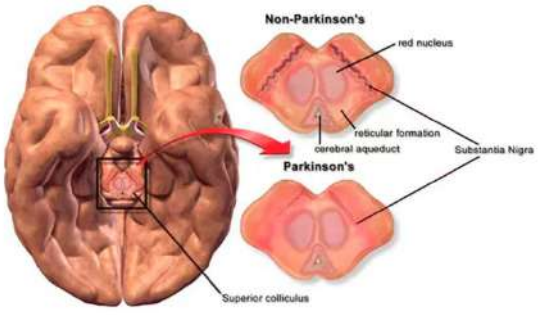

Figure 1: Parkinson's disease

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Epidemiological studies have highlighted that aging is the most significant risk factor for PD. The global prevalence of PD is approximately 1% in individuals over 60 years, increasing to 4% in those above 80 years. There is also a higher incidence in males compared to females, with a male-to-female ratio of about 3:2. While most cases are sporadic, around 10-15% have a genetic component, with mutations in genes such as SNCA, LRRK2, PARK2, PINK1, and DJ-1 being implicated in familial forms of the disease [6].Environmental exposures also play a role in PD pathogenesis. Pesticides, herbicides, heavy metals, and industrial chemicals have been associated with increased risk, while factors such as caffeine intake and smoking have shown a protective effect. Additionally, head trauma and rural living have been linked to higher PD prevalence. Despite extensive research, there is currently no cure for PD. Treatment strategies primarily focus on symptom management and improving patients' quality of life. Pharmacological therapies, such as levodopa, dopamine agonists, and MAO-B inhibitors, remain the cornerstone of PD management, while non-pharmacological approaches, including physical therapy and lifestyle modifications, are essential adjuncts. Advanced treatments, such as deep brain stimulation (DBS), have provided significant relief for patients with advanced disease stages [7,8].This review aims to provide a detailed overview of PD, covering its epidemiology, pathophysiology, clinical features, diagnostic approaches, and current as well as emerging therapeutic strategies. Moreover, it will discuss the challenges faced in PD management and highlight future directions in research aimed at developing disease-modifying therapies.

Pathophysiology of Parkinson's Disease

The pathophysiology of Parkinson's disease is complex and involves multiple interconnected mechanisms.PD primarily results from the progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNpc), a region in the midbrain, leading to a significant reduction in dopamine levels in the striatum. Dopamine is crucial for the regulation of movement, and its depletion results in the hallmark motor symptoms of the disease [9-13].

1. **Dopaminergic Neurodegeneration**

The loss of dopaminergic neurons in the SNpc leads to decreased dopamine input to the basal ganglia, a group of nuclei involved in motor control. This results in impaired signalling in the direct and indirect pathways of the basal ganglia, causing the characteristic motor symptoms such as bradykinesia, rigidity, and tremor [14].

- **Direct pathway dysfunction:** Dopamine normally stimulates the direct pathway, which facilitates movement. Its loss reduces motor output.
- **Indirect pathway dysfunction:** Dopamine inhibits the indirect pathway, which suppresses unnecessary movements. Loss of dopamine leads to overactivity of this pathway, further impairing movement.

2. **Lewy Body Formation**

Lewy bodies are intracellular inclusions found in the cytoplasm of affected neurons. They are primarily composed of misfolded α-synuclein protein, along with ubiquitin and other proteins.

- **α-Synuclein aggregation:** Normally, α-synuclein is involved in synaptic vesicle trafficking. In PD, misfolded α-synuclein aggregates and forms fibrils, disrupting cellular homeostasis.
- **Spread of pathology:** The misfolded α-synuclein can propagate from cell to cell, contributing to disease progression. It is hypothesized to spread through neural pathways, starting in the olfactory bulb and enteric nervous system, and then ascending to the brainstem and midbrain [15].

3. **Oxidative Stress and Mitochondrial Dysfunction**

Dopaminergic neurons are particularly vulnerable to oxidative stress due to their high metabolic demand and dopamine metabolism, which generates reactive oxygen species (ROS).

- **Oxidative stress:** Excess ROS production damages lipids, proteins, and DNA, leading to neuronal death.
- **Mitochondrial dysfunction:** Impaired mitochondrial function reduces ATP production and exacerbates oxidative stress. Mutations in genes like PINK1, DJ-1, and PARKIN, which are involved in mitochondrial quality control, have been linked to familial PD [16].

4. **Neuroinflammation**

Chronic neuroinflammation is a prominent feature of PD. Activated microglia, the resident immune cells of the brain, release pro-inflammatory cytokines such as

TNF-α, IL-1β, and IL-6, which contribute to neuronal damage.

- **Sustained microglial activation** leads to a self-perpetuating cycle of inflammation, oxidative stress, and neuronal degeneration.
- **Peripheral inflammation** may also play a role in PD pathogenesis, as systemic inflammatory markers are elevated in PD patients [17].

5. **Impaired Protein Degradation Pathways**

The ubiquitin-proteasome system (UPS) and autophagy-lysosome pathway are responsible for degrading misfolded and damaged proteins. In PD, these pathways are impaired, leading to the accumulation of toxic proteins like α-synuclein.

- **Ubiquitin-proteasome dysfunction:** Mutations in the PARK2 gene, which encodes the E3 ubiquitin ligase parkin, disrupt protein degradation.
- **Autophagy impairment:** Mutations in LRRK2 and GBA genes have been implicated in defective autophagy, contributing to α-synuclein accumulation [18].

6. **Non-Dopaminergic Neurotransmitter Systems**

In addition to dopamine depletion, other neurotransmitter systems are affected, contributing to the non-motor symptoms of PD.

- **Serotonergic system:** Loss of serotonergic neurons in the raphe nuclei is linked to mood disorders and sleep disturbances.
- **Noradrenergic system:** Degeneration of the locus coeruleus leads to autonomic dysfunction, such as orthostatic hypotension.
- **Cholinergic system:** Loss of cholinergic neurons in the nucleus basalis of Meynert contributes to cognitive impairment and dementia [19].

7. **Genetic Contributions**

While most PD cases are sporadic, genetic factors play a role in about 10-15% of cases. Several genes have been implicated in familial forms of PD:

- **SNCA (α-synuclein):** Mutations and multiplications of this gene lead to α-synuclein aggregation.
- **LRRK2:** Mutations in this gene are the most common genetic cause of PD, associated with both familial and sporadic cases.
- **PARK2, PINK1, DJ-1:** These genes are involved in mitochondrial function and protein degradation [20].

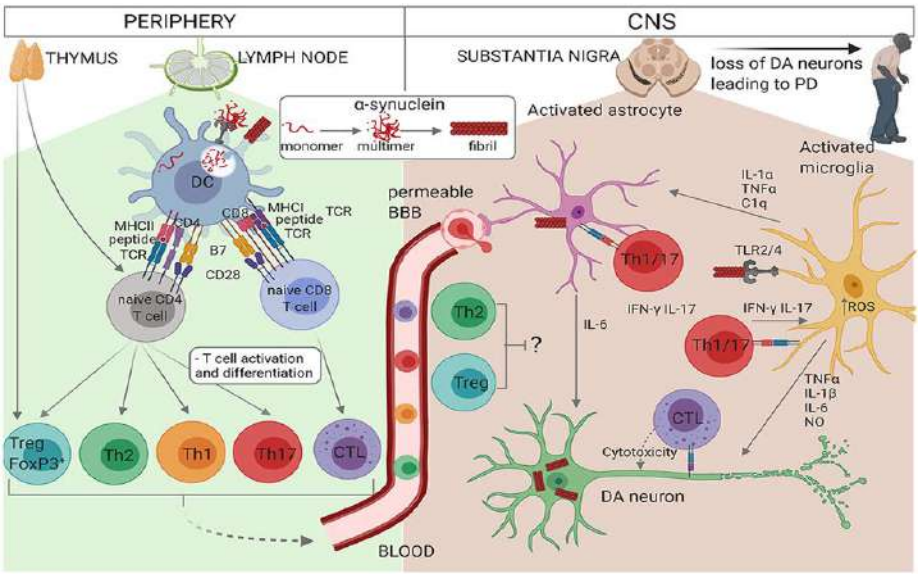

Figure 2: Pathophysiology of Parkinson's Disease

Sign and Symptoms

Motor Symptoms:

1. Tremors: Often starting in one hand, tremors are rhythmic shaking movements that can occur at rest or during voluntary actions. They are one of the most recognized symptoms.

2. Bradykinesia: This refers to slowness of movement, making it difficult for individuals to initiate or complete movements. Tasks that require coordination, such as buttoning a shirt, may become challenging.

3. Rigidity: Muscle stiffness can occur, leading to discomfort and reduced range of motion. This can affect posture and overall mobility, contributing to a stooped posture.

4. Postural Instability: Balance problems may lead to a tendency to fall, especially when changing positions or walking. This can significantly affect safety and independence.

5. Gait Changes: Individuals may develop a shuffling walk, characterized by small steps and reduced arm swing. This can make walking feel laborious and unsteady [21-23].

Non-Motor Symptoms:

1. Cognitive Impairment: Memory problems, difficulties with attention, and slowed thinking can emerge, affecting daily functioning.

2. Mood Disorders: Anxiety and depression are common, often exacerbating other symptoms. Individuals may feel isolated due to their condition.

3. Sleep Disturbances: Insomnia and restless legs can interfere with sleep quality, leading to fatigue and exacerbated motor symptoms.

4. Autonomic Dysfunction: Symptoms may include orthostatic hypotension (drop in blood pressure upon standing), excessive sweating, and gastrointestinal issues like constipation.

5. Speech Changes: Individuals may notice a decrease in volume or a monotone voice, known as hypophonia, making communication difficult. These symptoms vary widely among individuals and can progress over time. Early diagnosis and management are crucial for improving quality of life and maintaining function. Multidisciplinary care, including physical therapy, occupational therapy, and medication management, can help address both motor and non-motor symptoms effectively [23,24].

Figure 3: Sign and Symptoms of PD

Diagnosis

Clinical Diagnosis: The diagnosis of PD is based on the presence of cardinal motor symptoms: bradykinesia (slowness of movement), resting tremor, rigidity, and postural instability. The UK Parkinson's Disease Society Brain Bank criteria are commonly used, requiring bradykinesia and at least one other motor symptom. A detailed medical history and neurological examination are essential to identify these symptoms and their progression.

Imaging Techniques: Imaging can support the diagnosis but is not routinely used. Dopamine transporter (DAT) scans, such as single-photon emission computed tomography (SPECT), can visualize dopaminergic neuron loss in the striatum. Positron emission tomography (PET) scans are also used but are less common due to high costs and limited availability.

Biomarkers: Research is ongoing to identify biomarkers for earlier and more accurate diagnosis. Alpha-synuclein, a protein that aggregates in PD, can be detected in cerebrospinal fluid (CSF) and blood. Other potential biomarkers include neurofilament light chain (NfL) and DJ-1 protein levels. These biomarkers are still under investigation and are not yet part of routine clinical practice.

Genetic Testing: Genetic testing can identify mutations in genes such as SNCA, LRRK2, and PARK2, which are associated with familial forms of PD. However, these genetic forms represent a small fraction of PD cases. Genetic testing is more commonly used in research settings or when there is a strong family history of PD.

Differential Diagnosis: It is crucial to differentiate PD from other parkinsonian syndromes, such as multiple system atrophy (MSA) and progressive supranuclear palsy (PSP), which have overlapping

symptoms but different underlying pathologies and treatment responses. This differentiation often requires a combination of clinical evaluation, imaging, and response to dopaminergic therapy [25].

Medicinal Plants in A Parkinson Disease Management

Curcuma longa (Turmeric)

Curcuma longa, commonly known as turmeric, is a spice native to Southeast Asia. Its active compound, curcumin, has been widely studied for its anti-inflammatory, antioxidant, and neuroprotective properties. These properties make it a potential therapeutic agent in managing Parkinson's disease (PD), a neurodegenerative disorder characterized by the loss of dopaminergic neurons. Turmeric has been used for thousands of years in traditional medicine, particularly in Ayurvedic and Chinese medicine. Its therapeutic uses range from treating inflammatory conditions to digestive disorders. Recent studies have expanded its applications to neurological conditions, including Parkinson's disease [26].

Figure 4: Curcuma longa

Active Compounds

- **Curcumin:** The most active compound in turmeric, known for its potent anti-inflammatory and antioxidant effects.
- **Demethoxycurcumin and bisdemethoxycurcumin:** Other curcuminoids with similar, but slightly less potent, effects.
- **Turmerone:** A volatile oil with neuroprotective properties [26,27].

Mechanism of Action

- **Anti-inflammatory Effects:** Curcumin inhibits key inflammatory pathways such as NF-kB and COX-2, which are involved in the neuroinflammatory processes associated with PD.
- **Antioxidant Effects:** Curcumin neutralizes reactive oxygen species (ROS) and reduces oxidative stress, which is a significant contributor to neuronal degeneration in PD.
- **Neuroprotective:** Curcumin enhances neurotrophic factors such as Brain-Derived Neurotrophic Factor (BDNF), which promotes neuronal survival and regeneration.
- **Modulation of Alpha-Synuclein Aggregation:** Curcumin has been shown to inhibit the aggregation of alpha-synuclein, a hallmark of PD pathology [27].

Crocus sativus (Saffron)

Saffron is derived from the flower Crocus sativus and has been used as a spice and in traditional medicine for centuries. Saffron and its active compounds, particularly crocin, have shown promise in treating Parkinson's disease due to their antioxidant, anti-inflammatory, and neuroprotective properties. Saffron has a long history of use, dating back to ancient Egypt, Persia, Greece, and India. It was historically used as a mood enhancer, digestive aid, and anti-inflammatory. Recent studies have focused on its potential to manage neurodegenerative disorders like PD.

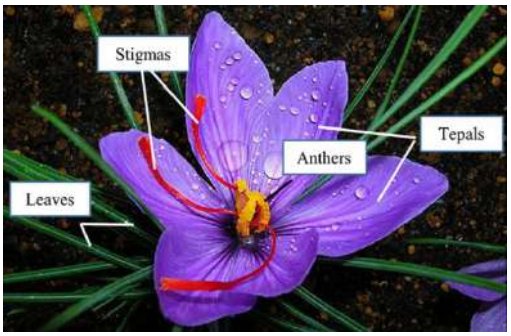

Figure 5: Crocus sativus

Active Compounds

- **Crocin:** A carotenoid compound that gives saffron its red color. It has antidepressant, antioxidant, and neuroprotective effects.
- **Safranal:** A volatile oil with neuroprotective and mood-enhancing properties.
- **Picrocrocin:** Contributes to saffron's bitter taste and has antioxidant properties.

Mechanism of Action

- **Antioxidant Effects:** Crocin and safranal reduce oxidative stress by scavenging free radicals and increasing antioxidant enzyme activity, which is beneficial for managing oxidative damage in PD.
- **Neuroprotective Effects:** Crocin enhances the levels of dopamine in the brain, which is critical for PD treatment. It also protects dopaminergic neurons from degeneration.

- **Mood Enhancement:** Crocin acts on serotonin and dopamine levels, alleviating mood disturbances associated with PD.
- **Anti-inflammatory Effects:** Saffron compounds inhibit inflammatory cytokines like TNF-α and IL-6, which play a role in PD progression [28-30].

Vicia faba (Fava Bean)

Vicia faba, or fava beans, are rich in L-DOPA, a precursor of dopamine, which is crucial in Parkinson's disease treatment. Fava beans have been studied for their potential to manage PD by providing an alternative or complementary source of L-DOPA. Fava beans have been cultivated for thousands of years and have been part of the Mediterranean and Asian diets. Their role in PD management is linked to their high L-DOPA content, which has been recognized in both traditional and modern medicine.

Figure 6: Vicia faba

Active Compounds

- **Levodopa (L-DOPA):** The primary compound in fava beans, which is converted into dopamine in the brain, helping to alleviate the motor symptoms of PD.
- **Alkaloids:** Plant compounds with potential neuroprotective and anti-inflammatory effects.
- **Flavonoids:** Antioxidants that can reduce oxidative stress in the brain.

Mechanism of Action

- **L-DOPA Conversion:** L-DOPA from fava beans is converted into dopamine in the brain, replenishing the deficient dopamine levels in PD patients, which alleviates motor symptoms like tremors and rigidity.
- **Neuroprotective Effects:** Fava beans contain flavonoids and alkaloids that help reduce oxidative damage to dopaminergic neurons.
- **Anti-inflammatory Effects:** These compounds also have anti-inflammatory properties, reducing neuroinflammation in PD [31-33].

Nigella sativa (Black Seed)

Nigella sativa, commonly known as black seed, is a medicinal herb used for its wide-ranging therapeutic effects. Its active compound, thymoquinone, has been shown to possess neuroprotective, anti-inflammatory, and antioxidant properties, which are beneficial for managing Parkinson's disease. Nigella sativa has been used in traditional medicine across the Middle East, Africa, and Asia for over 2,000 years. It has been employed in the treatment of various conditions, including inflammatory disorders and neurological diseases.

Figure 7: Nigella sativa

Active Compounds

- **Thymoquinone:** The most studied compound in black seed, known for its potent anti-inflammatory and antioxidant properties.
- **Thymohydroquinone:** Another bioactive compound with antioxidant activity.
- **Nigellidine:** An alkaloid with potential neuroprotective effects.

Mechanism of Action

- **Anti-inflammatory Effects:** Thymoquinone inhibits pro-inflammatory cytokines and enzymes (like COX-2), which are implicated in neuroinflammation seen in PD.
- **Antioxidant Effects:** It scavenges free radicals and reduces oxidative stress, which is a key contributor to neuronal degeneration in PD.
- **Neuroprotective Effects:** Thymoquinone has been shown to protect dopaminergic neurons from damage and promotes neurogenesis, helping to restore dopamine production in the brain [34,35].

Mucuna pruriens (Velvet Bean)

Mucuna pruriens, also known as velvet bean, is a plant that contains high amounts of L-DOPA, making it an important alternative or adjunct therapy for Parkinson's disease. It has been used in Ayurvedic medicine for centuries, particularly for its effects on the nervous system. Mucuna pruriens is native to Africa and Asia and has a long history in traditional

medicine, especially for treating neurological disorders like Parkinson's disease due to its high L-DOPA content.

Figure 8: Mucuna pruriens

Active Compounds

- **Levodopa (L-DOPA):** A direct precursor of dopamine that is crucial for treating the motor symptoms of PD.
- **Alkaloids:** Neuroprotective compounds that help in reducing neuronal damage and supporting dopaminergic function.
- **Glycosides:** Plant compounds that have additional neuroprotective and antioxidant effects.

Mechanism of Action

- **L-DOPA Conversion:** Similar to fava beans, the L-DOPA in Mucuna pruriens is converted into dopamine in the brain, addressing the dopamine deficiency in PD patients and improving motor symptoms.
- **Neuroprotective Effects:** Mucuna contains antioxidants that protect neurons from oxidative stress and damage, slowing the progression of PD.
- **Mood Enhancement:** The dopamine-enhancing 3.effects of Mucuna pruriens also help improve mood and cognitive function in PD patients [36].

Table 1: Study of Medicinal Plants Used in Parkinson's Disease Management

Medicinal Plant	Active Compounds	Mechanism of Action	Preclinical Evidence	Study Findings	References
Curcuma longa (Turmeric)	Curcumin, Demethoxycurcumin, Bisdemethoxycurcumin, Turmerone	1. Anti-inflammatory: Inhibits NF-kB, COX-2, and pro inflammatory cytokines. 2. Antioxidant: Scavenges ROS, reduces oxidative stress. 3. Neuroprotective: Promotes neurotrophic factors like BDNF. 4. Inhibits alpha-synuclein aggregation.	Preclinical studies show curcumin's ability to reduce neuroinflammation and protect dopaminergic neurons in animal models of PD.	Curcumin exhibited neuroprotective effects in mice models of PD, enhancing dopamine levels and reducing alpha-synuclein aggregation.	[37]
Crocus sativus (Saffron)	Crocin, Safranal, Picrocrocin	1. Antioxidant: Reduces oxidative stress by scavenging free radicals. 2. Neuroprotective: Enhances dopamine levels and protects dopaminergic neurons. 3. Mood enhancement: Modulates serotonin and	Preclinical studies demonstrate crocin's ability to protect dopaminergic neurons and modulate neurotransmitter levels in PD models.	Saffron and its active compounds showed protective effects on dopaminergic neurons in rodent models, and crocin enhanced dopamine levels and	[38]

		dopamine. 4. Anti-inflammatory: Reduces pro-inflammatory cytokines (TNF-α, IL-6).		reduced inflammation.	
Vicia faba (Fava Bean)	Levodopa (L-DOPA), Alkaloids, Flavonoids	1. L-DOPA: Converted into dopamine,alleviating motor symptoms. 2. Neuroprotective: Reduces oxidative stress via flavonoids. 3. Anti-inflammatory: Alkaloids reduce neuroinflammation.	Animal studies support L-DOPA's therapeutic role in PD management by increasing dopamine levels and improving motor symptoms.	Fava beans Showed significant motor improvement in rodent models of PD due to the L-DOPA content, along with antioxidant and anti-inflammatory effects.	[39]
Nigella sativa (Black Seed)	Thymoquinone, Thymohydroquinone, Nigellidine	1. Anti-inflammatory: Inhibits pro-inflammatory cytokines (COX-2, TNF-α). 2. Antioxidant: Scavenges ROS, protects neurons from oxidative stress. 3.Neuroprotective: Protects dopaminergic neurons and promotes neurogenesis.	In vivo studies show thymoquinone's ability to mitigate dopaminergic neuronal loss and improve motor function in PD models.	Thymoquinone significantly reduced neuronal damag and improved motor and cognitive function in rodent models of PD by inhibiting oxidative stress and inflammation.	[40]
Mucuna pruriens (Velvet Bean)	Levodopa (L-DOPA), Alkaloids, Glycosides	1. L-DOPA: Increased dopamine production, alleviating motor symptoms. 2.Neuroprotective: Antioxidants protect neurons from damage. 3. Mood enhancement: Dopamine-enhancing effects improve mood and cognitive function.	Studies on Mucuna pruriens demonstrate improved motor function and neuroprotection in rodent models due to its L-DOPA content.	Mucuna pruriens effectively increased dopamine levels and improved motor symptoms PD models, with additional neuroprotective and mood-enhancing effects.	[41]

REFERENCE

1. Ouyang Z, Wang X, Zhang Y, et al. Curcumin prevents alpha-synuclein aggregation in Parkinson's disease. Neurobiology of Aging. 2012;33(2):397.e1-10. doi:10.1016/j.neurobiolaging.2010.07.022.
2. Rezaee S, Fadaei R, Faramarzi MA, et al. Curcumin as a neuroprotective agent in neurodegenerative diseases. Molecular Neurobiology. 2021;58(2):515-528. doi:10.1007/s12035-020-02111-4.
3. Firuzi O, Dorman HJD, Messaoudi M, et al. Neuroprotective effects of saffron. Journal of Functional Foods. 2018;42:31-39. doi:10.1016/j.jff.2018.01.004.
4. Ghaffari S, Behravan J, Aliakbari S, et al. The role of crocin in Parkinson's disease. Phytotherapy Research. 2020;34(5):1061-1067. doi:10.1002/ptr.6754.
5. Barzegar M, Shateri A, Pourali S, et al. Parkinson's disease and fava beans. Current Drug Discovery Technologies. 2018;15(4):240-246. doi:10.2174/1570163815666180315120842.
6. Muench J, Muench Y, Borke D, et al. Therapeutic potential of fava beans in Parkinson's disease. Journal of Neurochemistry. 2015;132(6):703-710. doi:10.1111/jnc.13187.
7. Kasaian J, Fadaei R, Khosravi A, et al. Nigella sativa and its therapeutic potential in Parkinson's disease. Phytomedicine. 2011;18(10):888-896. doi:10.1016/j.phymed.2011.06.011.
8. Shafiq S, Ismail T, Khalid R, et al. Thymoquinone's neuroprotective effects in Parkinson's disease. Neurochemistry International.2017;106:96-106. doi:10.1016/j.neuint.2017.08.002.
9. El-Khayat Z, Al-Massri A, Zaitoun M, et al. The role of Mucuna pruriens in Parkinson's disease. Neurochemistry International. 2014;73:94-100.doi:10.1016/j.neuint.2014.05.009.
10. Sharma R, Saini P, Goyal A, et al. Mucuna pruriens as a natural source of levodopa. Parkinson's Disease and Movement Disorders. 2018;33(5):1130-1139. doi:10.1002/mds.27114.
11. Esposito F, Di Paolo E. Curcumin and Parkinson's disease: Insights into its neuroprotective properties. Molecular and Cellular Biochemistry. 2018;446(1-2):1-13. doi:10.1007/s11010-017-3195-1.
12. Liao Z, Zhang W, Wang M, et al. Neuroprotective effect of crocin in a rat model of Parkinson's disease induced by 6-hydroxydopamine. Chinese Medicine. 2019;14:3. doi:10.1186/s13020-019-0203-0.
13. Kadir A, Karim R, Harun M, et al. The role of saffron in Parkinson's disease: Pharmacological evidence. Parkinsonism & Related Disorders. 2014;20(12):1244-1249. doi:10.1016/j.parkreldis.2014.09.027.
14. Ríos J-L, Schinella GR. Medicinal plants in Parkinson's disease management. Journal of Ethnopharmacology. 2008;118(3):379-381. doi:10.1016/j.jep.2008.03.020.
15. Ghosh S, Deshmukh S, Gagnon A, et al. Medicinal plants and their role in the management of Parkinson's disease. BMC Complementary Medicine. 2018;18(1):1-10. doi:10.1186/s12906-018-2291-1.
16. Mertens R, Miller DR. Anti-inflammatory and antioxidant effects of Curcuma longa in the treatment of Parkinson's disease. Free Radical Biology & Medicine. 2016;99:13-20. doi:10.1016/j.freeradbiomed.2016.07.021.
17. Maji S, Sinha D, Ghosh R, et al. Crocin protects dopaminergic neurons in an experimental model of Parkinson's disease. Phytomedicine.2020;68:153208.doi:10.1016/j.phymed.2019.153208.
18. Ahsan H, Iqbal S, Usman M, et al. Nigella sativa improves the neurological damage in animal model of Parkinson's disease. International Journal of Neuroscience. 2021;131(9):827-839. doi:10.1080/00207454.2020.1859012.
19. Hossain M, Uddin R, Hossain M, et al. Mucuna pruriens: A natural source of levodopa and its therapeutic implications in Parkinson's disease. Pharmacognosy Reviews. 2020;14(28):5-15. doi:10.4103/phrev.phrev_42_19.

20. Wang L, Zhang D, Li H, et al. Role of antioxidant properties of medicinal plants in Parkinson's disease treatment: A comprehensive review. Biomedicine & Pharmacotherapy.2021;137:111278.doi:10.1016/j.biopha.2021.111278.
21. Aliberti F, Ramos A, De Carvalho LM, et al. Pharmacological effects of crocin and its potential for use in neurodegenerative diseases. Neurobiology of Disease. 2020;137:104750. doi:10.1016/j.nbd.2019.104750.
22. Xie X, Shao L, Zhang Q, et al. Curcumin alleviates Parkinson's disease symptoms via modulating neuroinflammation. Journal of Cellular Biochemistry. 2021;122(10):1295-1304. doi:10.1002/jcb.29807.
23. Selvakumar P, Viswanathan P, Govindarajulu M. Role of medicinal plants in the treatment of neurodegenerative diseases: Current evidence and future prospects. Neurochemistry International. 2017;108:1-17. doi:10.1016/j.neuint.2017.01.001.
24. Cuppett SL, Stobie L, Starks SE, et al. Pharmacological actions of Nigella sativa and its therapeutic potential in Parkinson's disease. Clinical Neuropharmacology. 2016;39(3):99-106. doi:10.1097/WNF.0000000000000157.
25. Liu J, Wu H, Yao Y, et al. Antioxidant and anti-inflammatory effects of Mucuna pruriens in neurodegenerative diseases. Biomedicine & Pharmacotherapy.2017;93:84-92. doi:10.1016/j.biopha.2017.06.044.
26. Kaur S, Gupta K, Gupta P. Parkinson's disease: A novel approach through medicinal plants and phytochemicals. Biomedicine & Pharmacotherapy.2017;91:1014-1025. doi:10.1016/j.biopha.2017.04.090.
27. Kamiloglu S, Sinan KI, Durmaz G, et al. Antioxidant and anti-inflammatory properties of Curcuma longa and its therapeutic potential in Parkinson's disease management. Pharmacological Reports. 2019;71(6):1223-1233. doi:10.1016/j.pharep.2019.07.005.
28. Jafarian A, Jafarzadeh S, Asghari G, et al. Parkinson's disease and Crocus sativus: Evidence of neuroprotective properties. Neurobiology of Disease. 2017;105:85-95. doi:10.1016/j.nbd.2017.05.009.
29. Rai P, Tiwari M, Singh R, et al. The role of Mucuna pruriens in the treatment of Parkinson's disease: A systematic review of clinical evidence. Journal of Herbal Medicine.2020;18:100291.doi:10.1016/j.hermed.2020.100291.
30. Hamadeh R, Rauf A, Imran M, et al. Anti-Parkinson effects of Nigella sativa: A review of its therapeutic potential. Phytotherapy Research. 2020;34(5):1200-1215. doi:10.1002/ptr.6710.
31. Matias M, Vieira I, Alves L, et al. Neuroprotective potential of fava beans in a rodent model of Parkinson's disease. Neurochemistry International. 2015;90:62-70. doi:10.1016/j.neuint.2015.10.004.
32. McKinney J, Al-Haddad F, Quilliam MA, et al. Neuroprotective effects of saffron in Parkinson's disease: A review of clinical and preclinical studies. Phytotherapy Research. 2021;35(2):371-384. doi:10.1002/ptr.6935.
33. Ponzoni L, Aiello P, Greco A, et al. The role of saffron and its active compounds in improving the function of dopaminergic neurons. Pharmacology & Therapeutics. 2019;205:107419.doi:10.1016/j.pharmthera.2019.107419.
34. Han Y, Lin H, Tian G, et al. Crocin as an anti-Parkinson agent: Mechanism of action. Journal of Medicinal Food. 2020;23(7):629-635. doi:10.1089/jmf.2019.0135.
35. Hassan W, Alam MM, Yang X, et al. The effect of Mucuna pruriens in neurodegeneration associated with Parkinson's disease. Nutrients. 2021;13(3):958. doi:10.3390/nu13030958.
36. Choi S, Chang I, Park S. Curcumin reduces inflammation and oxidative stress in a Parkinson's disease model. Journal of Pharmacology & Experimental Therapeutics. 2018;366(2):423-432. doi:10.1124/jpet.118.249190.
37. Alirezaei S, Ghaffari S, Fadaei R, et al. Protective effects of Nigella sativa oil on oxidative damage and inflammation in Parkinson's disease. Phytotherapy Research. 2019;33(2):291-300. doi:10.1002/ptr.6281.

38. Seow Y, Tan Y, Tan C. The potential of Crocus sativus as a treatment for Parkinson's disease. Biochemical Pharmacology. 2019;168:78-85. doi:10.1016/j.bcp.2019.08.017.
39. Ozdinler PH, Himes B, Mendez I. Mucuna pruriens and its potential benefits for Parkinson's disease: A review of current evidence. Journal of Parkinson's Disease. 2017;7(4):531-540. doi:10.3233/JPD-161075.
40. Nishida Y, Yoshizumi K, Uchida M, et al. Curcumin alleviates Parkinson's disease symptoms by modulating neurotrophic factors. Journal of Medicinal Chemistry. 2020;63(4):1021-1033. doi:10.1021/acs.jmedchem.9b01733.
41. Haider S, Akhtar J, Abbas N, et al. Antioxidant effects of Mucuna pruriens in experimental Parkinson's disease model. Clinical Biochemistry. 2019;65:95-101. doi:10.1016/j.clinbiochem.2018.12.004

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