

Murraya Koenigii Beyond Culinary Use: Advanced Neuroprotective and Anti-Parkinson Potential

Koyyana Yochanasree¹, Kunda Daniel Raju¹, Gummadi Veda Priya², K. Surya Neeraja Nagalla³, Dharmasoth Rama Dev^{4*}, R. Vasudhar³, P.Yamini¹ and D.Vasudha⁵

¹Department of Pharmacology, Vignan Institute of Pharmaceutical Technology, Duvvada, Visakhapatnam, Andhra Pradesh, India 530049

²Department of Pharmacognosy, Aditya College of Pharmacy (A), Surampalem, Andhra Pradesh, India 533437

³Department of Pharmacology, Vijaya Institute of Pharmaceutical Sciences for Women, Vijayawada, 521108.

^{4*}Department of Pharmacognosy, Vignan Institute of Pharmaceutical Technology, Duvvada, Visakhapatnam, Andhra Pradesh, India 530049

⁵Department of Pharmaceutical Chemistry, Vignan Institute of Pharmaceutical Technology, Duvvada, Visakhapatnam, Andhra Pradesh, India 530049

*Corresponding Author: ramajoy90@gmail.com

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ABSTRACT

The curry leaf (*Murraya koenigii* (L.) Spreng), well-known for its contribution in South Asian cuisine. But, recent studies show it has considerable pharmacological potential in neuroprotection and against neurodegenerative diseases. Background: Parkinson's disease (PD) is a progressive neurodegenerative disease which involves the loss of dopaminergic neurons and accumulation of α -synuclein. The current PD treatments are limited because they only alleviate symptoms and don't slow the disease process. Natural products are becoming more popular due to their low side effects and more concentrated process. *M. koenigii* is rich in bioactive phytochemicals particularly with carbazole alkaloids like mahanine, girinimbine and koenimbine, which are largely known as high antioxidants with anti-inflammatory and anti-apoptotic properties. These compounds impact on important molecular pathways linked to pathobiology of Parkinson's disease (PD) such as oxidative stress, mitochondrial dysfunction and neuroinflammation. Experimental research in in vitro and in vivo neurotoxicity models (rotenone and paraquat) have shown that it is capable of maintaining antioxidant defenses, enhancing behavioral outcomes, and enhancing dopaminergic neuron protection. Furthermore, *M. koenigii* can inhibit the aggregation of α -synuclein or acetylcholinesterase activity, in most of the cases, it can be prevented by diet. While preclinical studies are encouraging, some obstacles like low bioavailability, variable extracts and insufficient clinical studies must be addressed before it can be translated. This review covers the phytochemistry, the neuroprotective mechanism and the anti-Parkinson potential of *M. koenigii*, and suggests future research directions on the development of *M. koenigii* as a novel neurotherapeutic agent.

Keywords: *Murraya koenigii*; Parkinson's disease; neuroprotection; carbazole alkaloids; neurodegeneration.

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INTRODUCTION

Existing treatments, such as levodopa and dopamine agonists, are primarily symptomatic and largely only treat PD symptoms, but are often limited by significant side effects, such as levodopa-induced dyskinesia.

Given the multifactorial pathogenesis of PD with oxidative stress, mitochondrial dysfunction, neuroinflammation, and protein aggregation, there is a growing emphasis on medicinal plants with multiple bioactive components and lesser toxicity.

Murraya koenigii (curry leaf) is an important medicinal plant extensively used in Ayurveda for the treatment of various ailments. Recent research indicates that it has a profound neuroprotective effect against neurodegenerative diseases, especially Parkinson's⁵.

Botanical Description and Ethnomedicinal Uses

Murraya koenigii is a member of Rutaceae family and is found in all of India, Sri Lanka and south east Asia. Ethnomedicinal importance of *M. koenigii* has been established in neurological disorders due to its traditional usage as an anti-inflammatory, anti-diabetic, gastrointestinal and cognitive stimulant⁹.

*Author for Correspondence: ramajoy90@gmail.com

The traditional medicinal uses of M. koenigii encompass its antioxidant, anti-inflammatory, antiapoptotic, and neuroprotective properties, which are largely attributed to the presence of its rich phytochemical profile of carbazole alkaloids, flavonoids, terpenoids, and phenolic compounds⁹. The plant is also regarded as a “medhya rasayana” or brain tonic in Ayurveda, owing to its traditional application for boosting memory and cognitive functions¹⁷.

Pathophysiology of Parkinson’s Disease

The abnormal misfolding of α -synuclein into Lewy bodies, which disrupts synaptic transmission, mitochondrial activity and protein degradation systems within the cell, is another key component of the pathogenic mechanisms of PD^{24,25}. Dopamine depletion also contributes to the disease process by causing apoptosis of dopaminergic cells.²⁶ Protein aggregation is also a major part of PD pathogenesis, leading to the formation of Lewy bodies^{24,25}. It is worth noting that several of these molecular processes have been linked to the abnormal accumulation of α -synuclein in the substantia nigra pars compacta. As dopamine-producing neurons in the nigrostriatal pathway continue to die, the resulting neurotransmitter imbalance leads to the typical motor and non-motor signs and symptoms of the disease²⁶. These shared pathological pathways collectively lead to the progressive neurodegeneration and clinical deterioration in Parkinson’s disease²⁷.

Neuroprotective Mechanisms of Murraya koenigii

Antioxidant Activity

The antioxidant activity of Murraya koenigii is remarkable as a result of its high concentration of carbazole alkaloids, flavonoids, and phenolic compounds, which are responsible for the disease prevention properties seen in the plant. This plant has been found to significantly inhibit the process of lipid peroxidation and increase the activity of endogenous antioxidant enzymes like glutathione, catalase and superoxide dismutase (SOD), which helps in protecting the neuronal cells from oxidative damage and in maintaining the viability of dopaminergic cells involved in Parkinson’s disease²⁰.

Anti-inflammatory Pathways

Another significant factor that plays a role in the degeneration of neurons in Parkinson’s disease is persistent neuroinflammation. Damage to the neurons is exacerbated by inflammatory mediators and cytokines that are released by the activated microglial cells. The anti-inflammatory properties of M. koenigii are also noteworthy in protecting against neurodegenerative diseases, as it has been shown to inhibit pro-inflammatory cytokines like tumor necrosis factor alpha (TNF- α), interleukin-1 beta (IL-1 β), and interleukin-6 (IL-6), as well as inflammatory signaling pathways such as the nuclear factor kappaB (NF- κ B)^{21,22}.

Anti-apoptotic Effects

One of the ways dopaminergic neurons are lost in PD is through programmed cell death (apoptosis). The research

shows that M. koenigii can control the apoptotic signaling pathways by balancing the expression of proapoptotic proteins like Bax and antiapoptotic proteins like Bcl-2 to reduce the death of the neuronal cells caused by mitochondria and boost the survival of neuronal cells to oxidative and toxic stress²³. The plant’s anti-apoptotic properties may help slow the neurodegenerative process and help maintain neuronal integrity.

Neurotransmitter Modulation

The positive effects of M. koenigii are also linked with its action on neurotransmitter systems, including dopamine metabolism, that is severely affected in Parkinson’s disease. Restoration of dopamine levels and improvement of dopamine neurotransmission have been suggested in experimental models, which may positively influence motor coordination, rigidity and alleviation of Parkinsonian symptoms. Furthermore, some of the phytoconstituents found in M. koenigii could have a beneficial effect on cognitive function and neuronal communication, adding to its therapeutic potential in neurodegenerative diseases.

Mitochondrial Protection

One of the hallmarks features of PD is mitochondrial dysfunction, which is closely linked to neuronal apoptosis and oxidative stress, and impaired ATP production. The bioactivity of the phytochemicals found in M. koenigii can help to maintain the integrity of mitochondria, stabilize mitochondrial membrane potential, decrease oxidative stress, and enhance cellular energy metabolism, which in turn can promote neuronal survival and function²⁵. M. koenigii may help to break the cycle of oxidative injury and neuronal degeneration that is often seen in PD.

Anti-Parkinson Potential

The Pathophysiology of Parkinson’s Disease

The pathogenesis of Parkinson’s disease (PD) is extremely complex and is associated with a cascade of events that interact, such as oxidative stress, mitochondrial dysfunction, neuroinflammation, apoptosis, and abnormal aggregation of α -synuclein proteins into Lewy bodies. When production of reactive oxygen species (ROS) is excessive, it also causes damage to lipids, proteins and nucleic acids, eventually leading to the death of neurons. Mitochondrial dysfunction also exacerbates the neuronal damage by reducing ATP production and increasing oxidative stress. Chronic activation of microglial cells and the secretion of inflammatory mediators like the tumor necrosis factor (TNF- α) and interleukin-1 (IL-1 β) promote neurodegeneration; the build-up of α -synuclein (α S) disturbs the clearance of intracellular proteins and the synaptic signaling. These pathological changes combine to cause a gradual progression of motor symptoms (tremor, rigidity, bradykinesia, and postural instability) and non-motor symptoms (cognitive impairment and sleep apnoea)²⁶.

EXPERIMENTAL MODELS

Experimental Parkinsonian models are extensively used to evaluate the neuroprotective efficacy of medicinal plants

and phytochemicals. In neurotoxin induced models of Parkinson's disease with rotenone and paraquat, *Murraya koenigii* has been shown to possess high levels of neuroprotective properties²⁷. Rotenone induced models mimic mitochondrial complex I inhibition and oxidative stress – mediated dopaminergic degeneration, while paraquat exposure generates ROS and neuronal apoptosis. The observed neuroprotective effects are related to the antioxidant, anti-inflammatory and antiapoptotic properties of *M. koenigii* extracts, which are mainly attributed to its carbazole alkaloids, flavonoids and phenolic compounds²⁸.

In Vitro Studies

The in vitro study with neuronal cell lines gives valuable insights into the mechanism of the neuroprotective activity of *M. koenigii*. The plant extracts also have neuroprotective effects against neurotoxin induced toxicity in neuronal cells by maintaining the integrity of the mitochondrial membrane and reducing the occurrence of oxidative stress²⁹. Bioactive compounds of *M. koenigii* modulate cell death pathways and have a stimulatory effect on the endogenous antioxidant system such as the activity of superoxide dismutase, catalase and glutathione. The results are very promising for the use of *M. koenigii* as an agent for the prevention of oxidative stress-mediated neuronal damage in the context of Parkinson's disease.

In Vivo Studies

The neuroprotective potential of *M. koenigii* has also been validated in several in vivo experimental models of Parkinson's disease, including 6-hydroxydopamine (6-OHDA)- and MPTP-induced animal models.³¹ These studies demonstrated that administration of *M. koenigii* extracts significantly improves motor coordination, locomotor activity, and behavioral performance in Parkinsonian animals³². Histopathological and biochemical analyses further revealed reduced dopaminergic neuronal loss in the substantia nigra along with decreased levels of oxidative stress biomarkers such as lipid peroxidation products.³³ In addition, treatment with *M. koenigii* restored endogenous antioxidant enzyme activity and preserved striatal dopamine levels, highlighting its multifaceted neuroprotective action. Collectively, these findings emphasize the therapeutic promise of *M. koenigii* as a potential natural intervention for Parkinson's disease and related neurodegenerative disorders.

Molecular Targets and Multi-Target Approach

α -Synuclein Aggregation

Lewy bodies are one of the major neuropathological features of Parkinson's disease (PD), consisting of aggregation and misfolding of α -synuclein. *Murraya koenigii* phytoconstituents, such as carbazole alkaloids and flavonoids, have marked anti-aggregation properties which inhibit fibril formation and decrease proteotoxic stress in neuronal cells. These compounds also maintain protein homeostasis and limit the oxidative damage that occurs as a result of α -synuclein accumulation and the subsequent degeneration of dopaminergic neurons^{32, 34}.

Acetylcholinesterase Inhibition

Cognitive dysfunction is often secondary to late Parkinson's disease and is linked with cholinergic imbalance. *M. koenigii* extracts have acetylcholinesterase (AChE) inhibitory activity that aids to increase the cholinergic neurotransmissions and cognitive performance. The plant contains flavonoids and carbazole alkaloids, which play a role in enhancing memory, protecting the brain, and boosting the activity of synapses, by blocking excess degradation of acetylcholine^{30, 33, 35}.

Dopaminergic Neuroprotection

The neuroprotective effect of *M. koenigii* is primarily due to its capacity to protect dopaminergic neurons from oxidative stress, inflammation, and mitochondrial dysfunction. Bioactive molecules like mahanine, girinimbine and koenimbine can inhibit the generation of ROS, the release of inflammatory mediators, and the activation of apoptosis signaling pathways such as Bax/Bcl-2 and activation of caspases. The mechanisms act together to keep dopamine levels in the striatum and to enhance the survival of neurons in experimental models of Parkinson's disease³⁴⁻³⁶.

PHARMACOKINETICS AND SAFETY PROFILE

Toxicological evaluation shows that *M. koenigii* is quite safe for use, even at therapeutic levels, with very few side effects in experimental studies³⁵. The plant has been found to be well tolerated in acute and sub-chronic toxicity studies. A few phytoconstituents are, however, poorly soluble in water, metabolized quickly and have low oral bioavailability, potentially limiting their clinical efficacy³⁶. In recent times, researchers have investigated various novel delivery systems, such as nanoformulations, liposomal delivery systems, and phytopharmaceutical technologies to enhance the stability, absorption, brain targeting, and therapeutic efficacy of bioactive compounds of *M. koenigii* in neurodegenerative disorders³⁷.

CURRENT LIMITATIONS AND RESEARCH GAPS

Despite promising preclinical evidence, several limitations restrict the clinical translation of *Murraya koenigii* in Parkinson's disease therapy. Most available studies are confined to in vitro and animal models, while well-designed human clinical trials remain scarce³⁹. Variability in phytochemical composition due to geographical origin, extraction methods, and plant processing also affects reproducibility and standardization of therapeutic outcomes. In addition, the precise molecular mechanisms underlying its neuroprotective effects are not fully understood, particularly regarding signaling pathways involved in mitochondrial protection, α -synuclein aggregation, and dopaminergic neuron survival³⁹⁻⁴¹. Furthermore, issues such as poor bioavailability, rapid metabolism, and limited blood-brain barrier penetration of certain phytoconstituents continue to challenge its pharmaceutical development⁴².

FUTURE PERSPECTIVES

Advanced nanotechnology-based drug delivery systems, such as nanoparticles, liposomes and nanoemulsions can

enhance bioavailability, brain targeting and sustained release of bioactive compounds⁴⁰, which is worth studying in the future. Comprehensive mechanistic studies by molecular docking, omics technologies and signaling pathway analyses are also required to understand the multitarget neuroprotective action of *M. koenigii*. Importantly, large-scale clinical trials are needed to establish efficacy, safety, optimal dose, and long-term therapeutic benefits in patients with Parkinson's disease⁴¹.

CONCLUSION

Murraya koenigii is a potential medicinal plant, rich in phytochemicals such as carbazole alkaloids, flavonoids and phenolic compounds, showing protective effects against Parkinson's disease. Experimental data show that the plant possesses antioxidant, anti-inflammatory, anti-apoptotic, mitochondrial protection and dopaminergic protection effects, which are mediated by different molecular mechanisms. It has been shown to inhibit oxidative stress, neuroinflammation, α -Syn aggregation and neurotransmitter imbalance, indicating its potential as a multi-target therapeutic agent for neurodegenerative diseases. Preliminary results are promising, but additional pharmacokinetic studies, formulation research and well controlled clinical trials are required to confirm its therapeutic application in the treatment of Parkinson's disease in humans.

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