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Review Article

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Cognitive Dysfunction in Parkinson's Disease: A Traditional Medicine Therapeutic Approach

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Abstract

Background: *Parkinson's disease (PD) is an incurable complex neurodegenerative disease pathologically described with a six-stage alpha-synucleinopathy. In stage 4 of the disease, the toxic alpha-synuclein reaches the hippocampus and causes a decline in one or more cognitive abilities, including memory, attention, speech, or visual-spatial ability. Cognitive dysfunction is increasingly recognized among the most important pre-motor symptoms associated with PD, which can severely affect the patient's quality of life. Furthermore, this pre-motor symptom of PD shares many common pathophysiological mechanisms with oxidative stress, cytotoxicity, or neuro-inflammation in specific individuals.*

Objective: *To highlight the mechanisms of selected traditional medicine therapeutic approaches underlying the prevention and/or treatment of PD associated with cognitive functions.*

Methods: *Popular traditional herbal compounds, herbal medicines, herbal formulations, or traditional medicine active ingredients that have been reported to have promising prospects for the prevention and/or cure of PD associated with cognitive deficits were reviewed, and their mechanism of action was pinpointed. Considering the wearing-off effects of most "Western" medicines, for each selected traditional medicine (plants/plant extracts, leaves, or roots), the beneficial bioactive compounds were reported.*

Results: *Bioflavonoid compounds among medicinal plants have a potential antioxidant capacity on selective dopaminergic neurons. Targeting bioactive compounds with selective affinity to the ultrastructure of the substantia nigra neurons, the cerebral cortex, or the hippocampus reduces mitochondrial damage and alleviates PD-associated with non-motor symptoms. Tetrandrine is a promising traditional therapeutic alternative for blocking the neurotoxic effects of conventional anti-PD drugs. The neuroprotective effect of Curcumin on dopaminergic neurons is related to its activity as an anti-inflammatory promoter in the expression of a sort of "enzyme" that accelerates the formation of substances that cause neuroinflammation in the brain of patients with PD. Medicinal plant extracts with anti- or pro-apoptotic effects are good candidates to significantly regulate the neuronal cell division cycle that prompts behavioral and clinical signs of PD.*

Conclusion: *Medicinal plant extracts may combat cognitive deficits in PD through the inhibition of oxidative stress, the regulation of mitochondrial dysfunction, the reduction of toxic excitatory compounds, the inhibition of neuro-inflammation, the inhibition of neuronal apoptosis, and the inhibition of abnormal protein aggregation in the brain.*

Keywords: Parkinson's Disease, Cognitive Dysfunction, Traditional Medicine, Prevention, Therapeutic Approach

1. Introduction

Parkinson's disease (PD), the second most common and complex neurological disorder, is recognized by a mixture of typical motor symptoms that include, but not exhaustive, akinesia, rigidity, bradykinesia, resting tremor, and non-motor symptoms such as cognitive or learning and memory impairment [1-4]. It is well established that the pathological changes in specific brain size/volume and areas (the basal ganglia, cerebellum, thalamus, hypothalamus, limbic system, locus coeruleus, prefrontal cortex, etc.) are mainly marked by the degeneration of dopaminergic neurons [2,5-8]. The incidence of PD has increased year by year, with a global prevalence of 7-10 million people affected by the disease [9]. For instance, in stages 3-6 of PD, although supplementing dopamine and/or reducing dopamine degradation is the most common therapeutic approach, clinical symptoms (tremor, bradykinesia, rigidity, etc.) are also treated with Levodopa, dopamine receptor agonist, monoamine oxidase B inhibitors, cholinesterase inhibitors, and other surgical approaches including pallidotomy and subthalamic deep brain stimulation [2,10,11].

However, although cardinal clinical (motor) symptoms that appear late in stages 1-2 of PD are debilitating for the patient, pre-clinical (non-motor) symptoms (nausea, constipation, headache, sleep disorder, cognitive or learning and memory deficits, dementia, etc.) are also critical for the quality of life of patients with early-onset PD [12-15]. To address this, one may compare the efficacy of conventional Western drugs (due to their large spectra of side effects mainly after long-term administration) to traditional therapeutic approaches available and affordable for the most vulnerable population in countries where people have lower financial income or revenue. The long-term side effects of Western medicines and most importantly the incapacity to cure the disease have prompted the search for a more safe and effective treatment approach for PD with associated disorders. Traditional medicine has been used for decades to treat diseases such as the tremor of hands and head, which is nowadays attributed to PD [16]. For instance, traditional treatment approaches for memory consolidation suggest the consumption of either a single specific herb or herbal formula, trunk, barks, or roots [16,17].

In general, traditional medicine is commonly used to improve/preserve cognition or learning and memory ability in some Asian countries such as Japan, Korea, and China [13]. However, although there is still no clear evidence showing a definite beneficial effect of these traditional treatments and drugs for patients with PD (due to the diversity of its etiology and complexity of symptoms), more investigations are necessary to uncover societal allegations and examine all evidence. To date, as the world is going through many health challenges most of them decimating the elderly population, traditional medicine seems to be a strong alternative for the prevention and/or cure of PD with associated disorders including cognitive dysfunctions. Therefore, considering cognitive dysfunction as a pathological mechanism that can accelerate PD pathogenesis, this review paper attempts to highlight and propose the mechanism of some popular traditional medicines (herbal compounds, herbal medicines, herbal formulations) or traditional medicine active ingredients easy to access that may prevent and/or treat PD associated with cognitive deficits (See Figure).

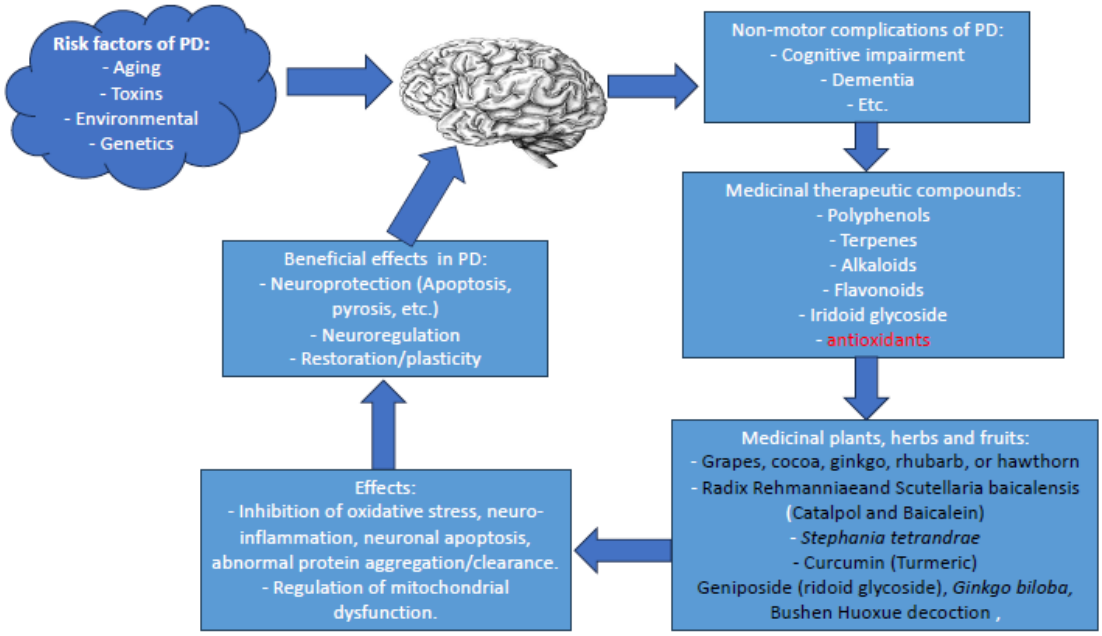

Figure: The Effects of Specific Medicinal Plants, Herbs, and Fruits in the Prevention of PD Associated with Cognitive Deficits

1.1. Traditional Medicines in the Inhibition of Oxidative Stress in Parkinson's Disease

It is generally accepted that the improvement of oxidative stress is among the most important pathophysiological mechanisms that drive the progression of PD [18-20]. In PD, oxidative stress results from the imbalance between the production (increased) and the clearance (elimination) of free radicals in specific brain areas. Any overproduction of free radicals in the brain may prompt neuronal cell death in PD pathogenesis [21]. For instance, it is commonly accepted that low molecular free radical scavenger glutathione (GSH) and free radical scavenging enzyme SOD, GSH-Px, in the substantia nigra of patients with PD are key signs of a state of oxidative stress [22-24]. In addition, elevated levels of ferric ion, lipid peroxide, hydroxyl, carbonyl, etc., are all biomarkers or contributors to cell death, which eventually accelerate neuronal apoptosis [25-27]. In traditional medicine, there is evidence of bioactive compounds in plant extracts, fruits, or leaves with potential antioxidant capacity [28]. For instance, a bioflavonoid compound such as Procyanidin, found in grapes, cocoa, ginkgo, rhubarb, or hawthorn, is known to be an effective natural antioxidant that can also significantly improve cognitive performance [29-31]. In fact, due to its fast absorption rate, Procyanidin can scavenge hydroxyl radicals, superoxide anion radicals and other active oxygen to reduce oxidative stress. Another study on a plant called *Clerodendranthus spicatus* also reveals that it is its flavonoid component that produces protective effects (antioxidant effects) on vulnerable dopaminergic neurons in PD [28].

Since flavonoids are known to have the ability to scavenge superoxide anion and hydroxyl radical, sometimes even better than vitamin C, it is possible that procyanidin or *Clerodendranthus spicatus* can significantly improve cognitive or learning and memory dysfunction, inhibits the surging of lipid peroxidation content (MDA), and the decrease SOD and GSH-Px activity in the substantia nigra of patients with PD [32-34]. An antioxidant activity coupled with the ability to clear out free radicals is indeed a good candidate with protective processes, including reducing neuronal cell damage, hence reducing the level of oxidative stress [28]. It is, therefore, possible that the prevention and treatment approaches against oxidative stress, which plays a critical role in the occurrence, progression, and deterioration of PD associated with cognitive dysfunction, may rely on screening the effectiveness of bioflavonoid compounds among medicinal plants that selectively have a potential antioxidant capacity on dopaminergic neurons.

1.2. Traditional Medicines to Regulate Mitochondrial Dysfunction in Parkinson's Disease

The pathophysiological mechanism of PD is centered on the abnormal morphology and function of mitochondrial cells induced by a toxic protein (alpha-synuclein) responsible for the mitochondrial genome and synaptic damage, the increased oxidative stress, the decreased membrane permeability, and ultimately, the appearance of cognitive impairment [35]. Considering mitochondria as cells that regulate the gene expression process and apoptosis in PD, there are also several lines of evidence suggesting a close relationship between mitochondrial dysfunctions, most neurodegenerative diseases and their associated disorders [35-39]. In fact, in early PD, it is known that the dysfunction of mitochondrial complex I plays a critical role in the progression of the disease [40]. For instance, it has been shown that the inhibition of the mitochondrial complex I reduces the production of cellular energy (ATP), causes lipid and protein degradation, or causes abnormal oxidative metabolic reactions, which will eventually lead to neuronal cell death in the substantia nigra [41,42]. It is, therefore, clear that both the overproduction of reactive oxygen species and the inhibition of the mitochondrial complex I are critical steps that aggravate the damage of the substantia nigra, hence, participate in PD pathogenesis. In recent years, some pharmacological targets of neuroprotection against neurodegenerative diseases and associated disorders include plant extracts or roots that contain compounds such as Catalpol and Baicalein, known to prevent mitochondrial dysfunction in the cerebral cortex and hippocampus [43-45]. For instance, Catalpol, one of the most important compounds in the Traditional Chinese Medicinal flowering plant called Radix Rehmanniae, has shown neuroprotective effects on mitochondrial damage by enhancing the activities of complex I and lowering the loss of mitochondrial membrane potential that drive PD symptoms associated with cognitive deficits [44].

In fact, Catalpol inhibits the NF-kB signaling pathway to reduce the microglia-mediated inflammatory response of neurons, thereby blocking the apoptosis of neural stem cells via the blood-brain barrier [46]. Concerning Baicalein, a flavonoid compound isolated from roots of Scutellaria baicalensis, a pre-treatment can greatly reduce mitochondrial damage and cell apoptosis by inhibiting reactive oxygen species production to preserve the mitochondrial membrane permeability and the eventual cytochrome C release to cytosol [43]. In the search for a new approach treatment for PD associated with cognitive deficits, it is possible that targeting these medicinal plant extracts with proven autonomic activities may prolong the incubation period of the disease and delay the clinical symptoms that usually reveal the advanced stage of PD. Overall, targeting bioactive compounds with selective affinity to the ultrastructure of the substantia nigra neurons, the cerebral cortex, or the hippocampus may reduce mitochondrial damage and alleviate PD associated with non-motor symptoms.

1.3. Traditional Medicine to Reduce Neurotoxicity in Parkinson's Disease

Among others, low levels of dopamine in the substantia nigra, striatum, or hippocampus are one of the most common features associated with PD [27,47,48]. It is, therefore, common knowledge that dysfunctions of specific brain neurotransmitters involved in movement or cognition may trigger a neurotoxicity process, which usually leads to neuronal cell death [22,49]. In fact, in addition to dopamine, it is common knowledge that dysfunctional neurotransmitters, such as glutamate, acetylcholine, gamma-aminobutyric acid, and encephalin, interact with each other in the

brain [50]. These interactions may result in neurotoxic effects when dopaminergic neurons are fully or partially degenerated [51]. For instance, it has been shown that when the extracellular glutamate levels reach a certain threshold, it damages or compromise the integrity of the hippocampus, one of the critical brain areas responsible for learning and memory or cognition [47,52,53]. In our search for novel treatment approaches to reduce neurotoxicity in PD-related cognitive deficits, one must not ignore the possible direct or indirect effects of traditional medicines on certain dysfunctional neurotransmitters.

Tetrandrine, a bisbenzylisoquinoline alkaloid extracted from the roots of *Stephania tetrandrae* is known to ameliorate cognitive or memory function [54,55]. It was shown that the co-administration of Tetrandrine and conventional anti-PD drugs markedly offset the possible oxidative neurotoxicity induced by most conventional PD drugs [40]. In fact, Tetrandrine consumption may decrease oxidative damage while blocking dopamine metabolism, it also enhances dopamine synthesis, increases tyrosine hydroxylase immunopositive neuronal survival, and eventually delays apoptosis of dopaminergic neurons in the brain [40,56]. A growing number of evidences suggests that Tetrandrine regulates the concentration of glutamate in the substantia nigra, cerebral cortex, and hippocampus, three of the critical brain areas involved in PD associated with cognition or learning and memory [52,53]. Tetrandrine may, therefore, offer a promising traditional therapeutic alternative for blocking the neurotoxic effects of conventional anti-PD drugs, hence only allowing the occurrence of their neuroprotective effects.

1.4. Traditional Medicine to Inhibit Neuroinflammation in Parkinson's Disease

Chronic neuroinflammation is a key contributor and one of the major pathophysiological mechanisms that fuels the significant cascade reaction in neuronal degeneration observed in PD [57]. As with almost all neurodegenerative diseases, a dysfunction of the neuroinflammatory response system implies activated microglia, the resident immune cells of the central nervous system [58]. Neuroinflammation is a response to neuronal damage where specialized defense cells (microglia) are tasked to rapidly remove the damaged cells by phagocytosis. Clinically, elevated levels of neuroinflammatory markers such as tumor necrosis factor -α (TNF-α), interleukin-1β (IL-1β), or interferon-γ (IFN-γ) strongly suggest that dysfunctional immune mechanisms produced in the dopaminergic neurons may potentially play a critical role in exacerbating PD pathology and apoptosis [59,60]. On the other hand, it has been reported that cognitive dysfunction in PD could be ameliorated by chemical-inhibited neuron damage [61,62]. In fact, recent research reported that a medicinal plant extract called Oxyphylla A can reduce both soluble and insoluble forms of α-synuclein-induced neuroinflammation [63,64]. This implies that the augmentation of the expression of neuroinflammatory markers will affect the nigrostriatal system of patients with PD, mainly in activated microglia, and the expression of neuroinflammatory factors will, therefore, positively correlate with the degeneration of dopaminergic neurons.

In recent years, Curcumin from Turmeric, a multifunctional plant known for its anti-inflammatory and neuroprotective effects, has been widely proposed to patients with PD and associated disorders, including cognitive deficits [65]. Curcumin is a potent antioxidant that scavenges reactive oxygen species and chelates toxic metals that cause neuroinflammation. Usually, it is the increased cytokine levels that trigger an inflammatory response to neuronal damage and eventually induce apoptosis by raising the level of nitric oxide (NO) in the brain and ultimately promote most neurodegenerative diseases [66,67]. Among these cytokines, IL-1β and TNF-α stand out as they are critical for macrophages and other cells to release other cytokines, such as IL-6 or IL-8, known to be dysfunctional in PD [68,69]. Curcumin may, therefore, act as a protective treatment approach on dopaminergic neurons in neuroinflammatory-induced PD associated with cognitive deficits via the inhibition of microglial cell activation, where the cascade of cytokine dysfunction takes off [70-73].

Furthermore, Curcumin may antagonize the loss of dopaminergic neurons in PD-associated cognitive deficits by decreasing the active oxygen content of dopaminergic neurons, hence the inhibition of inflammation [74]. The mechanism might be related to the up-regulation of cytokine expression, thereby inhibiting the inflammatory reaction and promoting the regeneration/protection of dopaminergic neurons [75]. Further, this may support the fact that the protective effect on dopaminergic neurons in PD-associated cognitive deficits is achieved by reducing the number of microglia and astrocytes in the substantia nigra and by down-regulating expression levels of protein and mRNA of inflammatory factors such as TNF-α [76]. Taken together, the neuroprotective effect of Curcumin on dopaminergic neurons may be related to its activity as an anti-inflammatory promoter in the expression of a sort of "enzyme" that accelerates the formation of substances that cause neuroinflammation in both the substantia nigra and the hippocampus of patients with PD.

1.5. Traditional Medicines that Inhibit Neuronal Apoptosis in Parkinson's Disease

Apoptosis, an active programmed cell death mechanism, is one of the most common pathologic processes of the central nervous system responsible for some core clinical and behavioral symptoms related to PD [77]. In Parkinsonism (e.g., PD with cognitive impairment), the efficacy of traditional medicines is therefore expected to protect dopaminergic neurons against factors-induced neuronal cell degeneration, including increased antioxidant substances, microglial activation, reduction of some inflammatory factors releases, modulate both protein and mRNA levels, which eventually prevent α -synuclein aggregation or non-clearance in the brain [78]. In fact, in some traditional medicines used for PD associated with cognitive deficits, there is evidence that the anti-apoptosis gene (e.g., Bcl-2, Bcl-xL, Bcl-w, Bcl-

1) and pro-apoptosis gene (e.g., Bax, Bak, Bad, Bid) are the important apoptosis-regulatory genes [79,80]. Geniposide, an iridoid glycoside compound extracted from the dried ripe fruit of Gardenia *jasminoides Ellis,* is known for their anti-apoptosis effects in alleviating inflammation responses caused by oxidative damage in neuronal cells [81,82].

Since in PD, it is accepted that suppressing apoptosis targeting dopaminergic neurons may also slow the progression of the disease, Geniposide may exert a neuroprotective effect on vulnerable dopaminergic neurons [83,84]. Clinically, there is evidence that Geniposide consumption may substantially increase the number of TH (thyroxine hydroxylase) positive neurons, which leads to reduced apoptotic neurons together with an improvement of abnormal behavior such as cognitive deficit commonly seen in PD [85]. The mechanism of action of Geniposide may, therefore, be closely related to the inhibition of neuronal apoptosis. This may justify why studies have shown that the early onset PD mainly depends on the integrity of the mitochondrial membrane to counter pro/anti-apoptotic factors [79,80,86,87]. Here, we further postulate that Geniposide can delay the apoptotic cascade in dopaminergic neurons. *Ginkgo biloba,* a large tree with fan-shaped leaves, is one of the most common herbal supplements also used to treat or prevent memory disorders [88]. The possible effects of *Ginkgo biloba* on brain disorders were pointed out by Wu and his team, where they have shown the neuroprotective effects of Ginkgo *biloba Pingchan* recipe on dopaminergic neurons [89].

Their study has clearly shown that in PD with its associated disorders, although a drug treatment of Ginkgo biloba Pingchan Recipe accelerated the proliferation of the neuronal cells, apoptosis was substantially plunged, and the expression levels of apoptosis-related gene vastly dropped [89]. They ended up by suggesting that *Ginkgo biloba Pingchan* recipe may regulate the cell division cycle by preventing fast cell growth and division or uncontrolled cell division, which is always the case in PD pathogenesis with associated disorders. Overall, in PD-induced apoptosis in dopaminergic cells, a medicinal plant extract with anti- or pro-apoptotic effects may be a good candidate to significantly regulate the neuronal cell division cycle by preventing fast cell growth and division, or uncontrolled cell division that prompts or behavioral and clinical signs of PD [90,91].

1.6. Traditional Medicines that Regulate Abnormal Brain Protein Aggregation in Parkinson's Disease

One of the key features of PD pathogenesis commonly recognized is the impaired degradation of misfolded and aggregated proteins in the substantia nigra [92-94]. It is also accepted that the abnormal formation/deposition/clearance of Lewy bodies (with its α-synuclein conformational change driving the death of the neuron) may lead to a substantial drop of the activity of the proteasome in the substantia nigra [95-97]. Furthermore, it is known that the overexpression and/or mutation of α-synuclein usually accelerates the dysfunction of mitochondrial cells and increases the sensitivity to oxidative stress to eventually promote neuronal cell death due to its cytotoxicity [97,98]. These interesting characteristics of PD pathogenesis show that the protein production system (protein deposition *per se*), or some related events when uncontrolled, are toxic to neurons [47].

Bushen Huoxue decoction, a Chinese herbal medicine, has proven its effectiveness in improving cerebral microcirculatory disturbance and treating cognitive impairment related to synapse density and antioxidant activities in the brain [90,99]. In fact, in PD with associated disorders, including cognitive deficits, it was shown that avoiding degeneration of dopaminergic neurons in the substantia nigra and formation/aggregation of many proteins such as α-synuclein, ubiquitin and their related enzymes in neurons may alleviate their toxic effects on neuronal cells [47,93,94,100]. It seems that Bushen Huoxue decoction may effectively restrain the oligomer of α-synuclein, which intensifies cell activity to eventually decrease apoptosis in dopaminergic cells and, therefore, may exhibit its neuroprotective effects in PD (a sort of protein misfolding disease). With the same postulate mentioned for the medicinal plant above, there is accumulating evidence of another Chinese patent medicine called Anchanling, in which the mechanism of action can be related to the improvement of protein function [25,93,94,101].

In fact, Anchanling is also a good candidate that can promote the degradation of α-synuclein, thereby reducing the accumulation/aggregation of intracellular proteins and the formation of inclusion bodies [102,103]. Taken together, screening any protein dysfunction in the brain may be of great significance for the prevention and treatment of PD with associated disorders as it can protect nerve cells by inhibiting the fibrosis process of α-syn protein or inhibit the oligomerization of α-syn and its cytotoxic effect on dopaminergic cells. Furthermore, the therapeutic mechanisms of most medicinal plants against PD with associated disorders, including cognitive deficit, may be closely related to up-regulation of specific protein expression (that can reinforce the effect of repairing nerve injury factors) and down-regulation of the specific protein expression (that can contribute to diminish the condensed expression of proteins) in dopaminergic cells.

2. Conclusion

Altogether, PD associated with cognitive deficits involves many interactions, factors, and mechanisms. To date, all Western conventional medical approaches to cure PD have been unsuccessful. However, it has become clear that traditional medicine may possess the advantages of multiple components and holistic regulation to be explored to naturally reduce, if not "cure" PD and its associated disorders. Many progresses have been made in the study of inhibition of oxidative stress, improvement of mitochondrial function or metabolism, resistance to neurotoxicity, and suppression of neuronal cell death by apoptosis. In this review paper, we have only highlighted some popular traditional medicinal plant extracts which show neuroprotective effects on

dopaminergic neurons (in substantia nigra) and showed beneficial improvements in PD associated with cognitive deficits through normal consumption. Although traditional medicine seems to have optimistic results in the treatment of PD with associated disorders, the complexity of most traditional medical plant extracts and the diversity of their bioactive ingredients remains elusive to ascertain their mechanism comprehensively. Therefore, while the existing traditional medical plant extracts used for PD must be deeply screened to better reveal their exact pharmacological effects and mechanisms of action, these new treatment approaches may be suggested to compensate for the shortage of conventional treatment approaches. Our study paved the way for research on selected medicinal plant PD-lowering therapies as alternative therapeutic interventions that can prevent the excitotoxicity related to neuroinflammation, protein misfolding or overaccumulation, and possibly the subsequent neurological sequels patients with PD with associated disorders may encounter.

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