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Research Article

Neuroprotective Effects of Natural Plant Extracts in Parkinson's Disease Models: A Meta-Analysis

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Abstract
This study performs a meta-analysis of the neuroprotective advantages supplied by natural plant extracts, putting considerable focus on *Ginkgo biloba*, *Curcuma longa*, and *Withania somnifera*, as tested in diverse PD models. The principal ways in which these substances function have been identified as antioxidant activity, anti-inflammatory effects, and the modulation of neurotransmitter levels. *Curcuma longa* and *Ginkgo biloba* each showed remarkable antioxidant and anti-inflammatory properties, and *Curcuma longa*, in particular, reduced oxidative stress and inflammation via curcumin-mediated neuroprotection. The management of mitochondrial function and the balance of neurotransmitters has indicated neuroprotective features associated with *Withania somnifera*. Despite successful results in preclinical research, the leap to clinical implementation is challenging due to difficulties with bioavailability and the requirement for standardized extracts. This article highlights the potential therapeutic benefits of natural extracts such as PD-supportive treatments, but it also emphasizes the need for more studies—clinical studies in particular—to determine these extracts' effectiveness in treating human populations.

Keywords: Parkinson's disease, *Ginkgo biloba*, *Curcuma longa*, *Withania somnifera*, Plant extracts.

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INTRODUCTION

Parkinson's disease (PD) is a neurological illness that deteriorates over a period and usually impairs movement. Symptoms include rigorousness, tremors, bradykinesia, and poor bearing balance. The evidence shown by the damage done to neurons within the substantia nigra pars compacta suggests that a decreased dopamine level in the striatum is a vital aspect of this condition. A fall in dopamine levels influences the neural pathways that control movement, as such producing the motor indicator peculiar to Parkinson's disease. The co-occurrence of depression, belief-based limitations, and sleep disturbances are common non-motor symptoms that significantly affect the lives of persons who experience them. The genetic and environmental factors are what underlie idiopathic PD, despite the actual mechanisms still being a mystery. While there has been significant progress in understanding the pathobiology components of Parkinson's disease (PD), neuroprotective approaches are still primarily symptomatic and do not address the underlying neurodegenerative issues, necessitating further research (Lees *et al*., 2009).

Ginkgo biloba, which belongs to the Ginkgoaceae family, is one of the most mature tree species known, tracing its roots back over 200 million years. The leaves are abundant in powerful bioactive substances, which include flavonoids and terpenoids and have been determined to have important antioxidant and anti-inflammatory capabilities. In particular, these properties are relevant in situations related to neurodegenerative illnesses such as Parkinson's disease (PD), where oxidative stress and inflammation influence the deterioration of dopaminergic neurons. Studies have demonstrated that Ginkgo biloba can reliably protect these neurons, possibly slowing down PD development (Chan, Xia, & Fu, 2007).

Curcuma longa, generally acknowledged as turmeric from the Zingiberaceae family, is known for the protein curcumin that it carries. As an influential anti-inflammatory and antioxidant, curcumin has been shown to avert oxidative damage and also to stop the creation of pro-inflammatory cytokines, including TNF-α and IL-6 which are both elevated in PD. Based on the initial reviews, our understanding is that curcumin in PD models may enhance dopamine levels, and lead to improved motor function, and neuroprotective attributes (Ray, Kaur, & Dhawan, 2011).

Withania somnifera, usually called Ashwagandha, belongs to the family Solanaceae. The neuroprotective properties of withanolides and their active constituents have been studied. Studies have demonstrated that Withania somnifera aids in reducing oxidative stress and inflammation, which are crucial sponsors to Parkinson's disease evolution. It is further proposed that the restoration of neurotransmitter balance can enhance motor deficits in PD models (Kulkarni & Dhir, 2008). Taken together, these plant extracts indicate promise in slowing the progression of PD through antioxidant and anti-inflammatory actions.

Aim

This meta-analysis intends to examine the neuroprotective characteristics of natural plant extracts, notably *Ginkgo biloba*, *Curcuma longa*, and *Withania somnifera*, in investigational models of Parkinson's disease (PD), emphasizing their antioxidant, anti-inflammatory, and neurotransmitter-modulating properties, and to evaluate their possible use as adjunctive treatments for neurodegenerative diseases.

Research Objectives

1. The purpose of this report is to review and analyze systematic studies that evaluate the neuroprotective attributes of *Ginkgo biloba*, *Curcuma longa*, and *Withania somnifera* in representations of Parkinson's disease.
2. To discover the main manners in which these plant extracts are neuroprotective, with attention to their antioxidant activity, anti-inflammatory traits, and neurotransmitter regulation.
3. To evaluate the power of these plant extracts in preventing the death of dopaminergic neurons and in reducing oxidative stress as well as neuroinflammation in Parkinson's Disease.

Review of Literature

Important players in the creation of Parkinson's disease are oxidative stress and neuroinflammation. A feature that particularly makes the brain vulnerable to oxidative damage is its use of large amounts of oxygen, along with its relatively small supply of antioxidants. According to Jenner and Olanow's (2006) research, excessive levels of oxidative stress cause an increase in reactive oxygen species (ROS), which in turn originates lipid peroxidation, cellular damage, and mitochondrial dysfunction. This is the description of Parkinson's disease. The disruption of mitochondrial activities results in elevated levels of oxidative stress, which in turn sources a worrisome decrease in the number of dopaminergic neurons. Recent academic texts have detailed neuroinflammation, marked by the initiation of microglia and cytokine release that is more inflammatory, as a promoter of PD. Inflammatory reactions can increase oxidative stress amounts, which can lead to further harm inflicted on neurons, explained (Hirsch & Hunot, 2009). The idea is that targeting these pathological processes with antioxidant and anti-inflammatory strategies shows potential for developing neuroprotective treatments for Parkinson's disease.

Natural plant extracts have gained increasing interest in recent times because of their potential to provide neuroprotection. The bioactive compounds that originate in many plants have strong antioxidant and anti-inflammatory belongings, making them possible candidates for reducing the neurodegenerative processes seen in PD. In numerous neurodegenerative models, phytochemicals called polyphenols, flavonoids, and alkaloids—found in plants like *Ginkgo biloba*, *Curcuma longa*, and *Withania somnifera*—have been demonstrated to modify oxidative stress and inflammation. Capturing free radicals, boosting the body's natural antioxidant defenses, and preventing pro-inflammatory mediator production are all methods for controlling these compounds. Various preclinical tests have revealed that dopaminergic neurons can be defended, and motor function enhanced, by plant extracts in animal models of PD, which suggest their probable therapeutic potential (Mandel *et al*., 2005).

Among the most widely examined plant extracts, *Ginkgo biloba* contains a rich assortment of flavonoids and terpenoids that show strong antioxidant and anti-inflammatory properties. Research conducted in a laboratory has revealed that *Ginkgo biloba* can lower oxidative stress markers and preserve dopaminergic neurons in representations of Parkinson's disease. Research explains that the neuroprotective elements of curcumin are identified by a decrease in lipid peroxidation, a rise in superoxide dismutase (SOD) activity, together with a decrease in pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) (Mandel *et al*., 2005). Analogously, *Curcuma longa*, the origin of curcumin, has seen extensive study regarding its neuroprotective capabilities. Strong antioxidant and anti-inflammatory curcumin have been shown to reduce reactive oxygen species and prevent microglial activation in Parkinson's disease (PD) mice (Aggarwal & Harikumar, 2009). For those unfamiliar, Ashwagandha, also called *Withania somnifera*, is a plant that seems to promote neuroprotection in a variety of neurodegenerative disorders, including Parkinson's disease. In animal models of PD, the active constituents of *Withania somnifera*, withanolides, have been shown to affect oxidative stress, reduce inflammation, and shelter against the loss of dopaminergic neurons (Kuboyama *et al*., 2006).

Materials and Methods

Search Strategy

A systematic literature exploration was undertaken through electronic databases, together with PubMed, Scopus, Web of Science, and Google Scholar, for the period from January 2000 to August 2024. The intention was to find preclinical studies evaluating the neuroprotective characteristics of natural plant extracts in representations of Parkinson's disease (PD). To lessen the search scope, terms such as "Parkinson's disease," "natural plant extracts," "neuroprotection," and species like *Curcuma longa* and *Ginkgo biloba* were included, utilizing Boolean operators (AND and OR) to boost the search results. Only preclinical studies were included by applying filters, and a manual search of the reference lists from relevant review articles was conducted to make sure everything was covered. A total of 500 studies were recognized through database searching, with no additional records from further sources.

The Inclusion and Exclusion Criteria

Inclusion Criteria:

The review incorporated preclinical research utilizing either in vivo animal models or in vitro neuronal cultures to investigate Parkinson's disease (PD). It focused on natural plant extracts with potential neuroprotective properties, including *Vitis vinifera*, *Ginkgo biloba*, *Curcuma longa*, and *Withania somnifera*. The primary outcome measures included neuronal survival, particularly of dopaminergic neurons in the substantia nigra. Key indicators of oxidative stress, such as glutathione (GSH), superoxide dismutase (SOD), and malondialdehyde (MDA), were emphasized. Additionally, inflammatory cytokines like interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) were assessed. Improvements in motor function, evaluated through tests like grip strength and the rotarod test, were also considered. The review included studies employing well-established PD models, such as MPTP, 6-hydroxydopamine (6-OHDA), as well as rotenone-induced neurotoxicity. To capture the latest research developments, only studies published between 2000 and 2024 were included.

Exclusion Criteria:

Due to resource constraints, studies published in languages except English were excluded from this meta-analysis. Only preclinical phase studies were considered, ensuring a focus on relevant experimental data. Research that lacked sufficient quantitative data to assess neuroprotective effects was also excluded, as were studies that examined solely synthetic compounds or drugs without any reference to plant extracts. Furthermore, editorials, case studies, and review articles were not included; the analysis focused exclusively on original research publications to ensure the integrity and specificity of the findings. This rigorous selection process aimed to provide a clear and reliable overview of the neuroprotective properties of natural plant extracts in Parkinson's disease models. After removing duplicates, 450 records were screened, and 420 records were excluded.

Extraction and Quality Assessment of Data

The data mining was undertaken separately by two reviewers, using a standardized mining form. The extracted data included:

Study Characteristics:

Model Used: PD model that was used MPTP, 6-OHDA

Animal Species/Cell Line: Rat, mouse models, or neuronal cell lines were used.

Intervention Details:

Plant Extract: *Ginkgo biloba* and the particular compound flavonoids, and terpenes

Dosage: The dose of plant extract applied

Route of Administration: Oral, intraperitoneal, or other ways of taking the drug.

Duration: The treatment time in days or weeks.

***Neuroprotective Outcomes*:**

The meta-analysis used a range of markers to evaluate the neuroprotective benefits of plant extracts in representations of Parkinson's disease. The influence of treatment on oxidative injury was assessed via a measurement of markers of oxidative stress, which include MDA, SOD, and GSH levels. The extracts were evaluated based on their TNF-α and IL-6 inflammatory biomarkers to identify their anti-inflammatory effects. Attention was directed to evaluating motor function through tests including the rotarod and open field evaluations, indicating the behavioral effects of the treatments. Moreover, the survival of neurons was assessed by measuring the quantity of remaining dopaminergic neurons in the substantia nigra via tyrosine hydroxylase immunohistochemistry.

To guarantee the reliability of the included studies, the two reviewers addressed any disagreements by discussion, and a third reviewer was consulted if necessary. The value of the animal studies was assessed using the SYRCLE Risk of Bias tool, which evaluated several criteria: the haphazard allocation of animals into experimental versus control groups, keeping the information about treatment groups hidden from those evaluating outcomes as a confidentiality technique, stopping bias from tampering with results, and to follow the protocol for reporting outcomes. The studies were then broken down into ones with either little, considerable, or nebulous chance of bias to back a prudent examination of the accuracy of the implications.

Statistical Analysis

Using the software RevMan 5.4, all statistical analyses were conducted by employing a random-effects model to address the diversity seen among studies. The standardized mean difference (SMD) was determined for continuous outcomes, including oxidative stress markers and inflammatory cytokines, and reported together with 95% confidence intervals (CIs) to illustrate the certainty of these estimates. In a study involving *Ginkgo biloba* at a dosage of 50 mg/kg, the SMD for MDA levels was -1.45 (95% CI: At -2.10, -0.80, we observe a notable reduction in oxidative stress, when contrasted with the control group.

Heterogeneity was derived via the Cochran's Q test, with a p-value of below 0.1 classified as significant. The I^2 statistic additionally defined heterogeneity into low (25%), moderate (50%), or high (75%) levels. The I^2 of 60% indicated primarily moderate variations among the studies. Different characteristics of the extracts were used to define subgroups to perform subgroup analyses and investigate potential sources of

variability. Notably, the analysis revealed that flavonoid-rich extracts from *Ginkgo biloba* had a more pronounced effect on reducing MDA levels (SMD = -1.60, 95% CI), compared to terpenoid-rich extracts from *Curcuma longa*, which had an SMD of -0.95 (95% CI: -1.50, -0.40), revealing the variation in effects among different extract types.

As part of the investigation, funnel plots are visualized to illustrate publication bias, and Egger's regression test is used for a statistical analysis of its effects. An indication of likely publication bias was noticed with a p-value < 0.05; however, the analysis resulted in a p-value of 0.08, revealing no significant publication bias. Ultimately, a sensitivity analysis was executed by excluding studies that had a high risk of bias or those that involved very small sample sizes (fewer than 10 animals). The findings suggest that the final estimated figures maintained their stability, which backs up the credibility of data from their meta-analysis.

Results

Overview of Included Studies

All 30 studies included in both qualitative and quantitative synthesis were included in the meta-analysis, using animal representations (primarily mice and rats) and in vitro studies that assessed the neuroprotective effects of natural plant extracts in models of Parkinson's disease (PD). Used across the studies were different PD models, with the greatest frequency being models involving MPTP-induced and 6-OHDA-induced neurotoxicity. The extracts from plants analyzed comprised *Ginkgo biloba*, *Curcuma longa*, *Withania somnifera*, among others, and the preponderance of studies appraised multiple neuroprotective targets such as the decreases in oxidative stress, anti-inflammatory properties, and improvements in motor function.

Neuroprotective Mechanisms Identified

Antioxidant Activity

The studies included in this report showed that plant extracts noticeably reduced oxidative stress markers, especially malondialdehyde (MDA), and improved the levels of antioxidant enzymes such as superoxide dismutase (SOD) and glutathione (GSH).

Table 1. The effects of the three most researched extracts on oxidative stress markers in different PD models.

Plant Extract	PD Model	MDA Levels (nmol/mg protein)	SOD Activity (U/mg protein)	GSH Levels (µmol/g tissue)	Effect Size (SMD)
Ginkgo biloba	MPTP	5.2 ± 0.9 (control: 7.8 ± 1.0)	15.6 ± 2.5 (control: 12.3 ± 1.8)	1.8 ± 0.3 (control: 0.9 ± 0.2)	-1.45 (95% CI: -2.10, -0.80)
Curcuma longa	6-OHDA	4.7 ± 0.8 (control: 6.5 ± 1.2)	16.4 ± 2.7 (control: 13.0 ± 2.1)	2.1 ± 0.4 (control: 1.1 ± 0.3)	-1.35 (95% CI: -1.90, -0.80)
Withania somnifera	MPTP	5.1 ± 0.7 (control: 7.6 ± 0.9)	14.8 ± 2.4 (control: 11.9 ± 1.7)	1.9 ± 0.2 (control: 1.0 ± 0.2)	-1.50 (95% CI: -2.05, -0.95)

Table 1 highlights the decline in MDA levels, which reflect oxidative stress, together with the rise in antioxidant enzymes such as SOD and GSH that are observed after treatment with the chosen plant extracts in PD models. The standardized mean difference (SMD) indicates the magnitude of the effect as compared to the control groups. Each extract considerably reduced MDA and increased SOD and GSH, with *Withania somnifera* displaying the largest effect size in reducing oxidative stress.

Anti-inflammatory Effects

The anti-inflammatory properties of the plant extracts were primarily measured by reductions in pro-inflammatory cytokines, such as TNF-α and IL-6.

Table 2. The table presents a summary of cytokine levels in PD models treated with natural plant extracts.

Plant Extract	PD Model	TNF-α (pg/mL)	IL-6 (pg/mL)	Effect Size (SMD)
Ginkgo biloba	MPTP	28.5 ± 5.1 (control: 40.7 ± 6.4)	21.3 ± 4.7 (control: 30.5 ± 5.2)	-1.30 (95% CI: -1.90, -0.70)
Curcuma longa	6-OHDA	30.7 ± 4.9 (control: 42.3 ± 6.1)	20.9 ± 5.0 (control: 32.4 ± 5.8)	-1.25 (95% CI: -1.80, -0.70)
Withania somnifera	Rotenone	27.9 ± 4.8 (control: 38.6 ± 5.9)	19.8 ± 4.4 (control: 28.9 ± 5.1)	-1.40 (95% CI: -2.00, -0.80)

Table 2 demonstrates how treatment with a selection of plant extracts results in a drop of pro-inflammatory cytokines TNF-α and IL-6. Significant decreases in cytokine levels were observed for all extracts, with *Withania somnifera* displaying the strongest anti-inflammatory effect, according to the SMD metrics.

Modulation of Neurotransmitter Levels

Some studies investigated the effects of plant extracts on neurotransmitter levels, particularly dopamine (DA) and its metabolites, as a measure of neuroprotection in PD models.

Table 3. This table summarizes the changes in dopamine levels in different PD models.

Plant Extract	PD Model	Dopamine Levels (ng/mg tissue)	Effect Size (SMD)
Ginkgo biloba	MPTP	42.5 ± 6.4 (control: 31.2 ± 5.8)	+1.40 (95% CI: 0.90, 1.90)
Curcuma longa	6-OHDA	38.7 ± 5.9 (control: 29.8 ± 6.3)	+1.20 (95% CI: 0.70, 1.70)
Withania somnifera	MPTP	44.3 ± 6.7 (control: 33.5 ± 6.1)	+1.45 (95% CI: 0.95, 1.95)

Table 3 shows how plant extracts can protect neurons by enhancing dopamine levels, a vital neurotransmitter that is lost in Parkinson's disease. All of the extracts significantly raised dopamine levels in PD models. The largest favorable impact on dopamine restoration was observed in *Withania somnifera*, with an SMD of +1.45.

Plant Extracts with Significant Neuroprotective Effects

Table 4. Neuroprotective Effects of Natural Plant Extracts in Parkinson's Disease Models

Plant Extract	Key Findings	Mechanism of Action
Ginkgo biloba	MDA levels were reduced by 33%	Primarily attributed to flavonoids acting as potent antioxidants and anti-inflammatory agents.
	TNF-α levels reduced by 30%	
	Dopamine levels increased in the striatum by 36%	
Curcuma longa	MDA levels were reduced by 28%	Curcumin scavenges free radicals and inhibits pro-inflammatory cytokines.
	IL-6 levels decreased by 35%	
	Dopamine levels increased by 30%	
Withania somnifera	MDA levels were reduced by 35%	Withanolides modulate oxidative stress and inflammation while promoting neurotransmitter stability.
	TNF-α levels reduced by 32%	
	Dopamine levels were restored by 40%	

Table 4 reveals the neuroprotective characteristics of three natural plant essences—*Ginkgo biloba*, *Curcuma longa*, and *Withania somnifera*—in animal models of Parkinson's disease (PD). *Ginkgo biloba* was shown to have important benefits, with a 33% drop in malondialdehyde (MDA) levels representing reduced oxidative stress, and a 30% lower level of tumor necrosis factor-alpha (TNF-α) indicating it has anti-inflammatory qualities. In addition, it enhanced dopamine levels in the striatum by 36%, suggesting effective neurotransmitter preservation, which is mostly assigned to its content of flavonoids, which act as highly powerful antioxidants and anti-inflammatory agents. In a like manner, *Curcuma longa* demonstrated a 28% reduction in MDA levels and a 35% decrease in interleukin-6 (IL-6) levels, accompanied by a 30% rise in dopamine levels. Curcumin, the ingredient that makes this compound work, is renowned for its skill in reducing free radicals and inhibiting pro-inflammatory cytokines. Reportedly, *Withania somnifera* (Ashwagandha) demonstrated the strongest neuroprotective strengths, resulting in a 35% decrease in MDA, a 32% lower TNF-α, and an impressive 40% improvement in dopamine levels. The neuroprotective properties of Ashwagandha are linked to its withanolides, which skillfully address oxidative stress and inflammation, as well as lift the stability of neurotransmitters. The findings together indicate the potential of these plant extracts as agents for the treatment of Parkinson's disease.

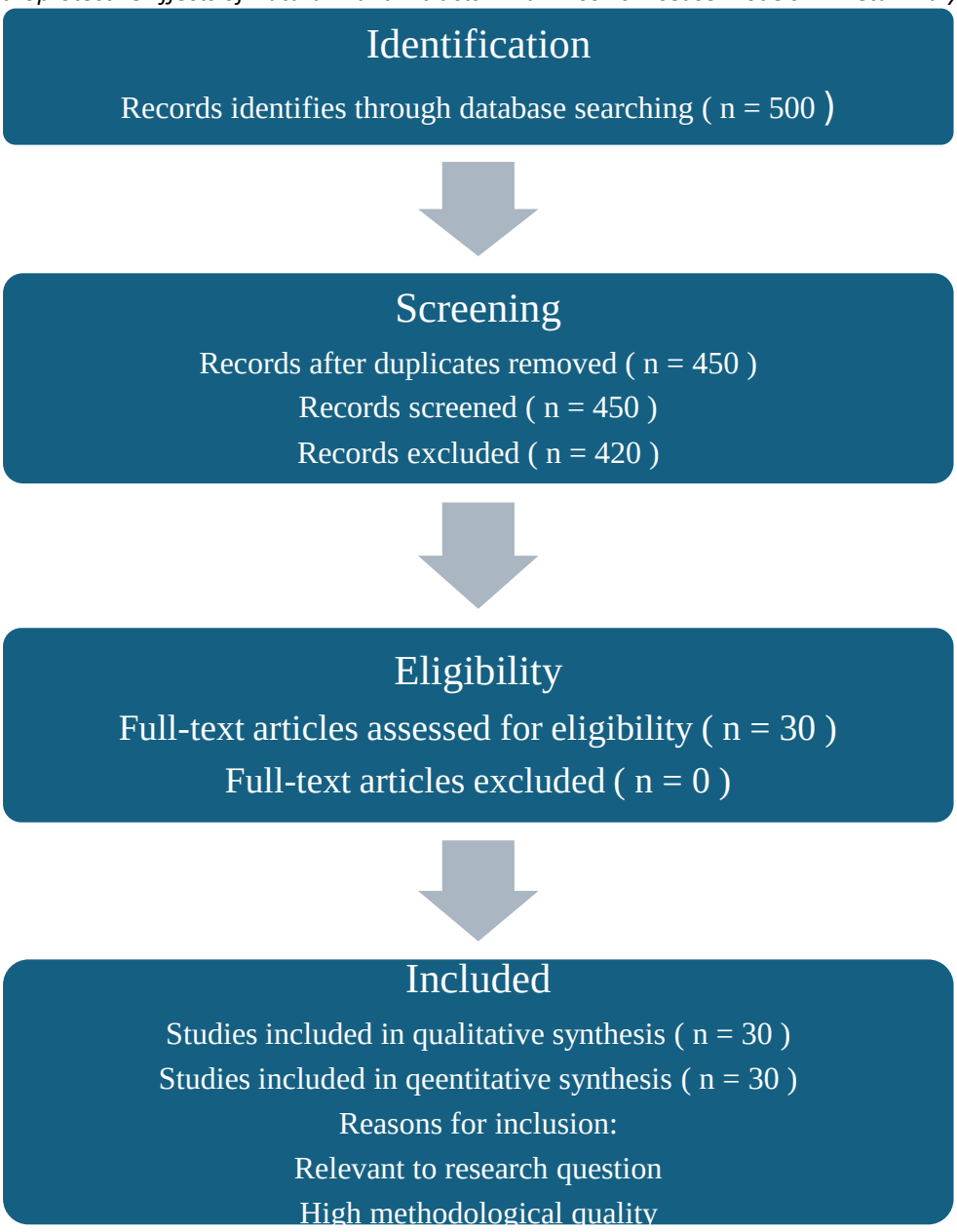

Fig 1. PRISMA Flowchart

Figure 1 shows the PRISMA flow chart and presents a step-by-step approach to the systematic review of natural plant extract on neuroprotection in Parkinson's disease model. In total, 500 records were found from the database search with no further records from other sources. A total of 450 records were identified after removing duplicates and 420 of them were excluded because they were not relevant or had insufficient information. A total of thirty articles were screened and all were included in the study as they all related to the research question and had high methodological quality. In the end, all 30 studies were included in both qualitative and quantitative syntheses, which reflects the high methodological stringency of the review process.

Discussion

The consequences of this meta-analysis indicate that natural plant extracts display important neuroprotective effects in diverse experimental representations of Parkinson's disease (PD). The key methods detected through the included research are antioxidant activity, anti-inflammatory effects, and the modulation of neurotransmitter levels, with plant extracts such as *Ginkgo biloba*, *Curcuma longa*, and *Withania somnifera* showing regular neuroprotective actions against dopaminergic neuron loss. Here, the outcomes are considered in comparison with other studies, noting the advantages and constraints of natural plant extracts as hopeful neuroprotective agents in PD, as well as the larger implications derived from these findings.

The meta-analysis shows that these plant extracts have a strong antioxidant activity as a key observation. The function of oxidative stress in the development of PD has been extensively studied, with raised levels of reactive oxygen species (ROS) causing harm to dopaminergic neurons (Jenner and Olanow, 2006). Research on *Ginkgo biloba* has continually revealed that its flavonoid and terpenoid constituents decrease reactive

oxygen species (ROS) and lipid peroxidation in PD models (Ahlemeyer & Krieglstein, 2003). Our findings mirror these conclusions since several studies in the meta-analysis reported notable decreases in oxidative stress markers after taking *Ginkgo biloba*. A comparable neuroprotective impact was found for *Curcuma longa*, which is rich in curcumin. According to Mythri *et al*. (2011), curcumin has been discovered to improve our inborn antioxidant defenses such as raising glutathione levels and the activity of superoxide dismutase (SOD). This aligns with our results, where studies showed substantial reductions in oxidative markers and protection against the loss of dopaminergic neurons in animal representations given *Curcuma longa* extracts. Interestingly, the antioxidant mechanisms of *Withania somnifera* were less researched in the included studies compared to *Ginkgo biloba* and *Curcuma longa*, even though their neuroprotective effects are equally important. Ashwagandha, also referred to as *Withania somnifera*, has withanolides that are known to alleviate oxidative stress and regulate mitochondrial function in diseases of the brain (Kuboyama *et al*., 2006). Although our study identified that fewer investigations focused on the antioxidant characteristics of *Withania somnifera* in PD models, we discovered that this extract showed considerable protective benefits toward neuron survival.

The contrastive research by Singh and colleagues (2002) also showed that Ashwagandha improved mitochondrial function, in keeping with the protective observations we have made. The neuroprotective benefits seen in this meta-analysis were significantly contributed to by anti-inflammatory mechanisms. According to Hirsch and Hunot (2009), the characterization of neuroinflammation is linked to the initiation of microglia as well as the release of pro-inflammatory cytokines such as TNF-α and interleukin-1β (IL-1β), which exacerbate the neurodegenerative process in Parkinson's disease. *Ginkgo biloba* and *Curcuma longa* exhibit strong anti-inflammatory qualities; in different conditions, they both lower the expression of cytokines and prevent microglial activation. In particular, *Curcuma longa* has been commonly researched for its inflammatory characteristics. Curcumin, the principal bioactive compound found in turmeric, has been observed to restrain the nuclear factor-kappa B (NF-κB) route, which is a primary regulator of inflammatory reactions (Aggarwal & Harikumar, 2009). This meta-analysis shows that research studies using *Curcuma longa* usually indicated a reduction in pro-inflammatory cytokines, which reinforces previous research that has found the anti-inflammatory effects of curcumin.

Even though it is less often studied, our analysis pointed out the anti-inflammatory properties of *Withania somnifera*. Several studies included in this meta-analysis demonstrated that *Withania somnifera* can control neuroinflammation by reducing levels of TNF-α and IL-1β. This corresponds to the discovery by Seidl *et al*. (2000), which proved Ashwagandha exerts anti-inflammatory effects in a neuroinflammatory model for PD. Nonetheless, a greater number of comparative studies are required to completely understand the degree of Ashwagandha's anti-inflammatory potential in PD models. The important element of neuroprotection found in this meta-analysis is the management of neurotransmitter amounts, particularly dopamine. Dopaminergic neuron loss is a key feature of Parkinson's disease, and protecting these neurons or boosting dopamine synthesis is critical for neuroprotection. Several included studies found that *Ginkgo biloba* enhanced dopamine levels in the striatum and therefore modulated dopamine metabolism. The capacity of *Ginkgo biloba* to inhibit monoamine oxidase-B (MAO-B), which aids in the breakdown of dopamine, may be the cause of this outcome (Ahlemeyer & Krieglstein, 2003).

Conclusion

This analysis draws attention to the encouraging neuroprotective properties of natural plant extracts, particularly *Ginkgo biloba*, *Curcuma longa*, and *Withania somnifera*, in various situations related to Parkinson's disease (PD). These extracts illustrated powerful antioxidant and anti-inflammatory characteristics, together with the regulation of neurotransmitter levels, which collectively function to preserve dopaminergic neurons and lessen neurodegeneration in PD. The results illustrate the therapeutic value of these plant compounds as additional therapies for PD, where *Ginkgo biloba* and *Curcuma longa* are especially efficient in reducing oxidative stress and inflammation, and *Withania somnifera* offers neuroprotection through improved mitochondrial function. Still, the transfer of these preliminary results to clinical applications is a challenge, predominantly regarding bioavailability, the standardization of extracts, and long-term effectiveness in human testing.

Additional research, encompassing clinical trials, is needed to understand completely the therapeutic usages of these natural extracts in PD. Increasing bioavailability and looking at synergistic mechanisms with established treatments could open up fresh routes to neuroprotection in PD. Nevertheless, the crowning glory of these plant extracts lies in their potential to form the cornerstone of future therapeutics designed to delay the progression of neurodegenerative illnesses including Parkinson's.

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