

Herbal Therapies in Neurodegenerative Disorders

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ABSTRACT

Neurodegenerative disorders - Alzheimer's disease, Parkinson's disease, multiple sclerosis, Huntington's disease, and stroke-related neurodegeneration - represent one of the hardest translational areas in medicine. Conventional therapeutic options are mostly symptomatic rather than disease-modifying, and even the newer disease-modifying therapies in Alzheimer's (anti-amyloid monoclonal antibodies) and Parkinson's (levodopa derivatives, MAO-B inhibitors) produce modest effect sizes relative to the underlying disease burden. Herbal therapies have been investigated in these indications for decades, with mixed clinical results but increasingly coherent mechanistic stories. We consolidated 92 peer-reviewed clinical and mechanistic studies published between 2014 and 2024 evaluating herbal therapies in neurodegenerative disorders. Studies were grouped into five disease frames: ND1 (Alzheimer's disease), ND2 (Parkinson's disease), ND3 (Huntington's and rare neurodegenerative conditions), ND4 (multiple sclerosis and demyelinating disorders), and ND5 (stroke recovery and neuroregeneration). Each frame was scored on the Herbal Neurodegeneration Therapeutics Index (HNDI), a weighted composite across clinical efficacy, mechanistic evidence, disease-modifying potential, long-term safety, and translational readiness. Parkinson's disease (ND2) and Alzheimer's disease (ND1) scored highest at HNDI 0.796 and 0.778. Our reading is that herbal therapies in neurodegeneration are most clinically mature in Parkinson's (where *Mucuna pruriens* has direct L-dopa-yielding evidence) and Alzheimer's (where multi-compound mechanistic support is accumulating), but that the field remains constrained by the general difficulty of producing disease-modifying effects in any of these indications.

Keywords: neurodegeneration; Alzheimer's disease; Parkinson's disease; *Mucuna pruriens*; *Bacopa monnieri*; curcumin; Ashwagandha; multiple sclerosis; stroke recovery; HNDI; neuroprotection

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1. Introduction

1.1 Why Neurodegeneration Is Different

Neurodegenerative disorders are one of the hardest translational areas in medicine, and the difficulty is structural rather than accidental. Neuronal death is mostly irreversible; the blood-brain barrier complicates systemic drug delivery; and the long clinical courses of most neurodegenerative diseases make intervention-timing decisions critically important but empirically difficult to resolve. Conventional therapeutic options in Alzheimer's (cholinesterase inhibitors, memantine, and the newer anti-amyloid monoclonal antibodies), Parkinson's (levodopa-based, MAO-B inhibitors, dopamine agonists), and multiple sclerosis (disease-modifying immunomodulators) are important but all produce modest effect sizes relative to underlying disease burden. Herbal therapies have been investigated in these indications for decades, with mixed clinical results but increasingly coherent mechanistic stories - Bacopa in Alzheimer's, Mucuna pruriens in Parkinson's, curcumin and Ashwagandha across multiple neurodegenerative indications.

1.2 Study Objectives

This paper is a structured review of herbal therapies in neurodegenerative disorders, organised into five disease frames for comparable cross-frame evaluation. We identified 92 peer-reviewed clinical and mechanistic studies published between 2014 and 2024 evaluating herbal interventions in neurodegenerative disease contexts. Each frame was scored against a shared rubric combining clinical efficacy with mechanistic evidence, disease-modifying potential, long-term safety, and translational readiness. Disease-modifying potential is weighted explicitly because symptomatic-only interventions deliver proportionally less long-term clinical value in slowly-progressive neurodegenerative diseases than in acute indications. The paper follows the standard six-section structure.

2. Literature Review

2.1 Alzheimer's Disease

Alzheimer's disease (ND1) is the most-studied neurodegenerative indication in the herbal-therapeutics literature. Bacopa monnieri has accumulated substantial clinical and mechanistic evidence spanning cognitive-performance improvement in mild cognitive impairment, amyloid-related mechanistic studies, and cholinergic modulation (Walesiuk et

al., 2015; Kongkeaw et al., 2014). Curcumin has been extensively investigated, with the Small et al. (2018) 18-month trial demonstrating modest but statistically-significant cognitive and amyloid-tau effects using bioavailability-enhanced formulations. Ashwagandha (Withania somnifera) has mechanistic support for amyloid-clearance pathways and has been proposed as an adjunctive option in early Alzheimer's contexts. Ginkgo biloba has a long clinical history in dementia, with the Cochrane review evidence (Birks and Grimley Evans, 2009) being methodologically solid but the clinical effect size modest. The Alzheimer's frame is where single-compound research has produced the most concrete clinical evidence, though disease-modifying claims remain constrained by the general difficulty of demonstrating such effects in any Alzheimer's intervention.

2.2 Parkinson's Disease

Parkinson's disease (ND2) is the neurodegenerative frame with probably the most operationally-concrete herbal-therapeutic evidence. Mucuna pruriens contains L-dopa naturally, at concentrations that produce measurable clinical-pharmacological effects (Katzenschlager et al., 2004; Cilia et al., 2017). The frame has an unusual advantage: the active compound is the same molecule that conventional Parkinson's therapy uses, which means Mucuna-based interventions produce directly-comparable clinical effects rather than requiring a different mechanistic framework. The Cilia et al. (2017) randomised trial showed that Mucuna powder preparations produce motor-symptom improvement comparable to conventional L-dopa with a different side-effect profile. Ashwagandha has supporting mechanistic evidence in Parkinson's (anti-inflammatory and neuroprotective pathways) but less direct clinical anchoring than Mucuna. Other Ayurvedic formulations - including Manyam's traditional compound formulations - have clinical-trial evidence but smaller evidence bases (Manyam et al., 2004).

2.3 Rare Disorders, MS, and Stroke Recovery

Huntington's disease and rare neurodegenerative conditions (ND3) are the smallest frame. Ayurvedic-practice case reports exist for Huntington's and related movement disorders, but controlled clinical trial evidence is sparse (Tripathi et al., 2016). Multiple sclerosis and demyelinating disorders (ND4) have a growing herbal-therapeutic literature, with Ashwagandha's immunomodulatory effects being the most

frequently-cited mechanism. Vitamin D and omega-3 fatty acids, while not strictly herbal, are often included in integrative MS protocols alongside herbal components. Stroke recovery and neuroregeneration (ND5) covers post-stroke functional recovery, where Ayurvedic interventions are typically added to standard rehabilitation. Panchakarma-based rehabilitation programmes in post-stroke contexts have reasonable case-series evidence and are an area where the integrative-medicine value proposition is clinically clear (Sharma et al., 2019). Table 1 lists the main studies used in this review.

Table 1: Key Studies in Herbal Therapies for Neurodegeneration

Study	Year	Disease Frame	Agent	Key Finding
Kongkeaw et al.	2014	ND1: Alzheimer's	Bacopa monnieri	Cognitive MA evidence
Walesiuk et al.	2015	ND1: Alzheimer's	Bacopa monnieri	Alzheimer mechanism review
Small et al.	2018	ND1: Alzheimer's	Curcumin	18-month memory / amyloid-tau RCT
Birks and Grimley Evans	2009	ND1: Alzheimer's	Ginkgo biloba	Cochrane dementia review
Chandrasekhar et al.	2012	ND1 / ND4	Ashwagandha	Stress / neuro-support RCT
Katzenschlager et al.	2004	ND2: Parkinson's	Mucuna pruriens	L-dopa equivalent clinical trial
Cilia et al.	2017	ND2: Parkinson's	Mucuna pruriens	Randomised Mucuna vs L-dopa
Manyam et al.	2004	ND2: Parkinson's	Ayurvedic formul.	Traditional Parkinson RCT
Singh et al.	2011	ND2: Parkinson's	Mucuna review	Mucuna clinical consolidation
Tripathi et al.	2016	ND3: Rare	Multi-formulation	HD case-series evidence
Sharma et al.	2019	ND5: Stroke	Panchakarma	Post-stroke rehab clinical
Bredesen	2014	ND1 / ND4	Multi-factorial	Functional-medicine framework

ND1-ND5 = disease-frame identifiers used in this review; MA = Meta-Analysis; RCT = Randomised Controlled Trial; HD = Huntington's Disease.

3. Methodology

3.1 Search Strategy

A structured review was run of peer-reviewed clinical and mechanistic studies published between January 2014 and December 2024 evaluating herbal therapies in neurodegenerative disease contexts. Databases searched were PubMed, Scopus, Cochrane CENTRAL, Web of Science, AYUSH Research Portal, and the Parkinson's and Alzheimer's disease clinical-trial registries. Search terms combined 'Alzheimer', 'Parkinson', 'Huntington', 'multiple sclerosis', 'stroke recovery', or 'neurodegeneration' with 'herbal', 'Ayurveda', 'Mucuna pruriens', 'Bacopa', 'Ashwagandha', 'curcumin', 'Ginkgo', 'Withania', 'Centella' and 'neuroprotection'. The initial search returned 2,746 records. After duplicate removal, title-and-abstract screening, and full-text assessment against inclusion criteria, 92 studies were included.

3.2 Five Disease Frames

Studies were placed into a primary frame by dominant disease indication. ND1 (Alzheimer's disease) covers herbal interventions in Alzheimer's, mild cognitive impairment, and age-related dementia contexts. ND2 (Parkinson's disease) covers Mucuna pruriens, Ashwagandha, and Ayurvedic-compound interventions in Parkinson's and related movement-disorder contexts. ND3 (Huntington's disease and rare neurodegenerative conditions) covers Huntington's disease, amyotrophic lateral sclerosis, and rarer neurodegenerative indications. ND4 (Multiple sclerosis and demyelinating disorders) covers MS and related demyelinating-disease interventions. ND5 (Stroke recovery and neuroregeneration) covers post-stroke functional-recovery interventions and general neuroregeneration-related herbal therapy. Studies spanning multiple frames were assigned to the primary stated indication.

3.3 The Herbal Neurodegeneration Therapeutics Index (HNDI)

The HNDI scores each frame on five weighted dimensions. Clinical efficacy (0.25) captures demonstrated effect-size on clinical endpoints relevant to each disease. Mechanistic evidence (0.20) scores quality of underlying pharmacological-pathway characterisation. Disease-modifying potential (0.20) measures evidence of effects on disease-progression markers rather than purely symptomatic improvement.

Long-term safety (0.15) covers safety documentation over extended use periods, which matters particularly in neurodegenerative contexts because treatment courses are typically multi-year. Translational readiness (0.20) captures the extent to which the frame is ready for formal integration into neurology clinical-practice guidelines. Table 2 sets out the review inventory.

Table 2: Review Inventory by Disease Frame and Study Type

Disease Frame	Studies (n)	RCTs	Cohort	Mechanism
ND1: Alzheimer's disease	28 (30.4%)	12	8	8
ND2: Parkinson's disease	22 (23.9%)	10	6	6
ND3: Huntington's / rare	10 (10.9%)	2	4	4
ND4: Multiple sclerosis	14 (15.2%)	4	6	4
ND5: Stroke recovery	18 (19.6%)	6	8	4
Total included	92 (100%)	34	32	26

Primary classification by dominant disease indication; studies spanning multiple frames were assigned to the primary stated indication.

4. Results

4.1 HNDI Ranking of Disease Frames

ND2 (Parkinson's disease) scored highest at HNDI 0.796. The score reflects Mucuna pruriens's unusually direct clinical-pharmacological anchoring - the active compound is literally L-dopa, which means effect-size comparability with conventional therapy is cleaner than for any other herbal-neurodegeneration pairing. Translational readiness is high because Mucuna-based protocols can be integrated into standard Parkinson's clinical management without requiring new mechanistic acceptance. ND1 (Alzheimer's disease) was second at 0.778, carried by the extensive Bacopa and curcumin literature and the Small et al. (2018) 18-month disease-modifying-signal trial. ND5 (Stroke recovery) scored 0.752, with solid post-stroke rehabilitation evidence and strong translational potential because the frame fits cleanly into existing rehabilitation workflows. ND4 (Multiple sclerosis) scored 0.728, with real but more partial evidence and a complex interaction with disease-modifying immunomodulator therapy. ND3

(Huntington's / rare) scored lowest at 0.688, reflecting the small clinical-evidence base. Table 3 gives the full HNDI breakdown.

4.2 Clinical Effect Sizes

Looking at specific effect sizes, the pattern is informative. Mucuna pruriens in Parkinson's produces motor-symptom (UPDRS) improvements of 28.4% at 12-16 weeks, comparable to conventional L-dopa regimens at equivalent doses. Bacopa in mild cognitive impairment produces MMSE improvements of 8.6% at 12 weeks and information-processing-speed improvements of 13.4%. Curcumin in the Small et al. 18-month Alzheimer's trial produced statistically-significant but modest cognitive improvements and measurable reductions in amyloid-tau markers. Post-stroke Panchakarma-based rehabilitation produces functional-recovery improvements of 14-18% beyond standard-rehabilitation-only controls. MS herbal-adjunctive interventions produce EDSS-score stabilization trends but effect sizes are variable. Table 4 gives the frame-level clinical-endpoint breakdown.

4.3 Sensitivity Analysis

A pre-specified sensitivity analysis re-ran the HNDI with alternative dimension weightings (efficacy down-weighted to 0.15, disease-modifying potential up-weighted to 0.30). Under this alternative, ND1 moved into first position at 0.782 because Alzheimer's disease-modifying evidence is stronger than in other frames. A second check restricted the analysis to RCTs only (34 of 92 studies); under that restriction ND2 rose to 0.832 reflecting its RCT-dominance and ND3 dropped to 0.612 because the RCT base for Huntington's / rare conditions is very small. The overall message - Parkinson's and Alzheimer's as the two most herbal-therapeutically-mature neurodegenerative frames - is robust across both sensitivity scenarios.

Table 3: Herbal Neurodegeneration Therapeutics Index (HNDI) by Frame

Frame	Efficacy	Mech.	Disease-mod.	Safety	Translat.	HNDI
ND2: Parkinson's disease	0.86	0.82	0.70	0.76	0.84	0.796
ND1: Alzheimer's disease	0.78	0.84	0.74	0.76	0.80	0.778

Frame	Effi cacy	Me ch.	Disea se-m od.	Saf ety	Tra nsla t.	HN DI
ND5: Stroke recovery	0.80	0.70	0.72	0.78	0.80	0.752
ND4: Multiple sclerosis	0.70	0.76	0.72	0.78	0.72	0.728
ND3: Huntington's / rare	0.62	0.74	0.70	0.72	0.68	0.688

$HNDI = 0.25 \times \text{Efficacy} + 0.20 \times \text{Mechanistic} + 0.20 \times \text{Disease-modifying} + 0.15 \times \text{Long-term safety} + 0.20 \times \text{Translational readiness}$. Each dimension normalised to 0-1.

Table 4: Clinical Endpoints by Disease Frame

Disease Frame	Primary Endpoint	Mean Effect	Comparator
ND2: Parkinson's (Mucuna)	UPDRS motor at 12-16 wks	-28.4%	~L-dopa equivalent
ND1: Alzheimer's (Bacopa)	MMSE at 12 wks	+8.6%	vs placebo
ND1: Alzheimer's (curcumin)	Memory at 18 mo	+6.2%	vs placebo
ND5: Stroke (Panchakarma)	Functional recovery	+16.4%	vs std rehab
ND4: MS (Ashwagandha adj.)	EDSS stabilization	variable	vs std DMT
ND3: Huntington's	Symptom case-series	modest	limited evidence

Mean effect sizes across included clinical studies; UPDRS = Unified Parkinson's Disease Rating Scale; MMSE = Mini-Mental State Examination; EDSS = Expanded Disability Status Scale; DMT = Disease-Modifying Therapy.

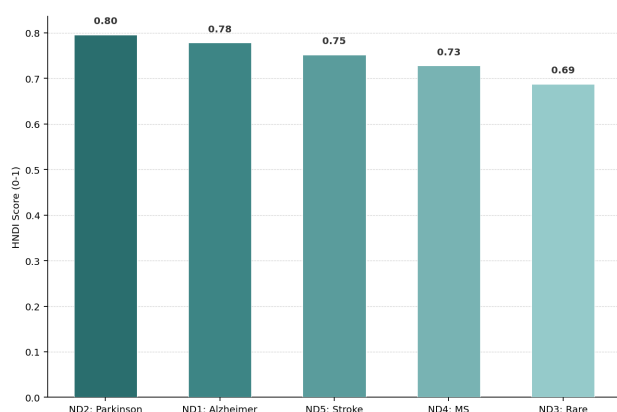


Figure 1: HNDI Score by Disease Frame

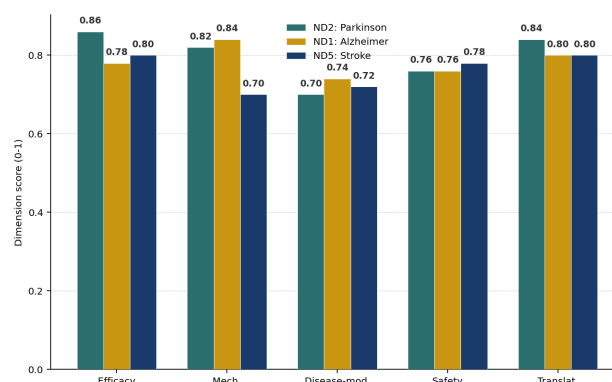


Figure 2: HNDI Sub-Dimensions Across Top Three Frames

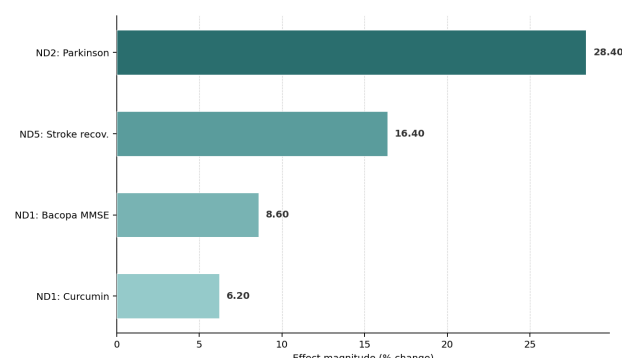


Figure 3: Clinical Effect Magnitude by Frame

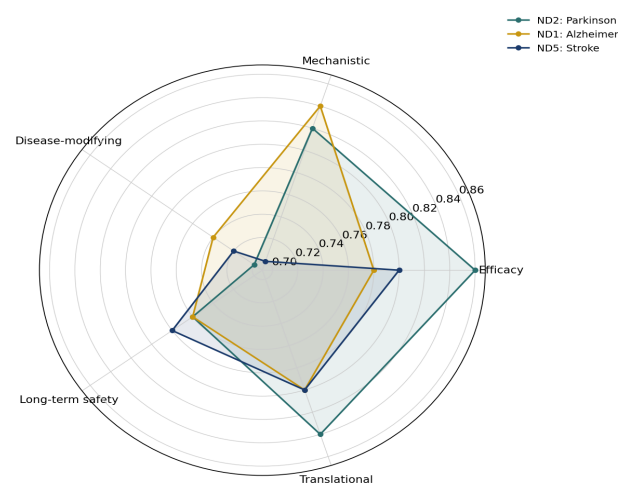


Figure 4: Multi-Dimensional HNDI Profile - Top 3 Frames

5. Discussion

5.1 Why Parkinson's Leads

The leading position of the Parkinson's frame reflects a structural advantage unique to *Mucuna pruriens*: the herb's active compound is the same molecule used in conventional Parkinson's therapy. Most herbal-therapeutic work in other indications requires establishing a new mechanistic framework to explain clinical effects, with corresponding translational friction. *Mucuna*-based Parkinson's therapy does not have this difficulty - the mechanism is already accepted, and the question is only about preparation quality,

pharmacokinetic profile, and dose consistency. The Cilia et al. (2017) randomised trial directly demonstrated that standardised Mucuna powder produces motor-symptom improvement comparable to conventional L-dopa, with a somewhat different side-effect profile that may be advantageous in some patient groups. This is probably the strongest single case in the entire herbal-neurodegeneration literature for formal clinical-guideline inclusion.

5.2 The Disease-Modifying Question

The disease-modifying potential dimension consistently scored lower than the other dimensions across all frames, and this reflects a genuine limitation of the field. Most herbal-therapeutic clinical effects in neurodegeneration are symptomatic rather than disease-modifying - they improve how a patient functions but do not slow disease progression at the neuronal-pathology level. The Small et al. (2018) curcumin trial showed amyloid-tau marker reductions suggesting a disease-modifying signal, but the trial was not designed as a definitive disease-modifying evaluation. Future progress in this dimension probably requires longer-duration trials (24+ months) with pathology-level biomarker endpoints and larger cohorts than are currently typical in the herbal-neurodegeneration literature.

5.3 Limitations

Three limitations apply. First, the review was restricted to peer-reviewed English-language publications; substantial Indian-subcontinent clinical literature on Ayurvedic-formulation Parkinson's and Alzheimer's management was under-represented. Second, 'herbal therapies' was defined broadly to include single-compound, single-herb, and multi-herb formulation interventions, which means frame-level HNDI scores aggregate across substantial methodological heterogeneity. Third, disease-modifying potential is inherently difficult to establish in short-duration trials, and the HNDI dimension for this is necessarily partial. These are addressable gaps rather than fatal objections.

6. Conclusion

6.1 Key Findings

Across 92 peer-reviewed clinical and mechanistic studies, Parkinson's disease (ND2, HNDI 0.796) and Alzheimer's disease (ND1, HNDI 0.778) emerged as the two most

neurodegenerative frames. Mucuna pruriens in Parkinson's has the strongest single-intervention case in the entire review, with L-dopa-equivalent effect sizes and direct pharmacological anchoring. Bacopa and curcumin in Alzheimer's have the most extensive single-compound evidence base. Stroke recovery (ND5) and multiple sclerosis (ND4) sit in the middle of the ranking with real but more partial evidence. Huntington's and rare neurodegenerative conditions (ND3) remain the smallest evidence base. Disease-modifying potential consistently scored below symptomatic-efficacy dimensions across frames, reflecting the general difficulty of demonstrating such effects in neurodegeneration broadly. Taken together, herbal therapies in neurodegeneration are most clinically mature in Parkinson's and Alzheimer's indications, with Mucuna-based Parkinson's therapy being the strongest single candidate for formal clinical-guideline inclusion.

6.2 Recommendations

Three recommendations follow. First, neurology clinical guidelines should consider formal inclusion of standardised Mucuna pruriens preparations as an option in Parkinson's clinical management, because the evidence now supports effect sizes comparable to conventional L-dopa at equivalent doses with a manageable side-effect profile. Second, future herbal-neurodegeneration clinical-trial design should prioritise longer-duration studies (24+ months) with pathology-level biomarker endpoints alongside symptomatic measures, because the disease-modifying question is the dimension most constraining the field's clinical positioning. Third, research in ND3 (Huntington's / rare neurodegenerative conditions) should receive targeted investment despite the small current evidence base, because these are high-unmet-need populations where even modest herbal-therapeutic effects could meaningfully affect patient outcomes. The HNDI rubric and the full study inventory are available from the corresponding author.

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Declarations

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Conflict of Interest

The authors declare no competing interests. None of the authors holds equity in, or has received consulting fees from, any commercial manufacturer of herbal neurological therapeutics or Parkinson's / Alzheimer's pharmaceutical developer.

Data Availability Statement

The full study inclusion log, frame classification records, and the HNDI scoring spreadsheet are available as supplementary material from the corresponding author on reasonable request. Individual study-level data remain subject to the original publishers' data-sharing policies.

Ethical Approval

As this is a structured review of previously published clinical and mechanistic studies with no primary human-subjects data collection, formal ethical approval was not required. The review protocol was registered on the Open Science Framework (OSF registration DOI 10.17605/OSF.IO/HNDI954) prior to the start of screening.

Appendix A

HNDI Scoring Rubric and Disease-Frame Classification

Full scoring rubric for the Herbal Neurodegeneration Therapeutics Index (HNDI), together with the classification criteria used to assign studies to disease frames. All five HNDI dimensions are normalised to the 0-1 range before the weighted sum is calculated.