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Neuropharmacological Investigations on Potential of Some Indian Medicinal Plants in the Integrative Management of Cognitive Disorders.

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ABSTRACT

Neurodegenerative diseases such as Alzheimer's and Parkinson's are characterized by progressive cognitive decline and impaired neuronal function. With growing concerns over the side effects and limitations of current synthetic drugs, there is increasing interest in exploring traditional herbal remedies. This study investigates the neuropharmacological potential of *Bacopa monnieri* (Brahmi), a well-known Ayurvedic herb, through pharmacogenetic evaluation, phytochemical screening, and chromatographic fingerprinting. The plant extract demonstrated the presence of key bioactive compounds, including bacosides, alkaloids, flavonoids, saponins, and tannins, which are known for their neuroprotective, antioxidant, and cognition-enhancing properties. Microscopic and physicochemical analyses confirmed compliance with standard identity and purity criteria. Thin Layer Chromatography (TLC) revealed a distinct bacoside-A band (Rf ≈ 0.54), validating chemical identity, while fluorescence analysis supported crude drug quality control. These findings provide a scientific basis for incorporating *Bacopa monnieri* into integrative therapeutic strategies for cognitive disorders, advocating for further in vivo and clinical evaluations.

Keywords: *Bacopa monnieri*, cognitive disorders, neuroprotection, Alzheimer's disease, phytochemical screening, herbal nootropics, bacosides, antioxidant activity, Ayurvedic medicine, chromatographic fingerprinting.

1.INTRODUCTION

Neurons use chemical and electrical signals to support the coordination of all fundamental aspects of life. When a neuron releases an electrical or chemical signal, it travels down its axon (a specialized projection) to the neighbouring cell. These signals can be retained by root-like dendrites. There are around 86 billion neurons in the human brain. Hence, a growing fetus generates approximately 250,000 neurons each minute (Fields et al., 2020; Heiney et al., 2021).

An enormous communication network is created because each neuron is connected to a thousand others. Neurons are the cells that make up the nervous system. Neurons are the cells in the brain responsible for transmitting and receiving signals(Duan et al., 2020a; Yang et al., 2020). Brain elements, including cognitive and motor neuron function, can be lost rapidly due to neurodegenerative illnesses, posing a significant problem for the elderly. Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), and amyotrophic lateral sclerosis (ALS) are neurodegenerative illnesses (Angelucci et al., 2019; Jensen et al., 2020).

Degeneration of the noradrenergic system is a pathological hallmark of many neurodegenerative diseases including Parkinson's disease, Alzheimer's disease, and Huntington disease. Indeed, pathology in the principal source of noradrenaline, the locus coeruleus (LC), can occur before the loss of other neurotransmitter systems commonly associated with such conditions or cerebral atrophy. Furthermore, the role of noradrenaline in diverse cognitive processes is well established, including vigilance, attention, and learning and memory. Yet, the degree to which noradrenergic systems contribute to the cognitive and behavioural changes resulting from neurological disease is often under-recognized despite early research reviews and more recent work to integrate the evidence into a coherent neurocognitive framework. (Negin Holland *et al.*2021). According to the U.S. National Institute on Aging, in the United States alone there are as many as four million cases of the most extreme form of cognitive breakdown, namely the dementia of Alzheimer's Disease.Other types of dementia add to the burden imposed on society by this tragic human affliction. For every case of diagnosed dementia there are probably several additional cases of individuals with Mild Cognitive Impairment (MCI) or ARCD (Age-Related Cognitive Decline) (Parris M. Kidd 1999).

Alzheimer's disease (AD) is a progressive, neurodegenerative disease that primarily affects the elderly population, and is estimated to account for 50–60% of dementia cases in persons over 65 years of age (Francis *et al.* 1999). The main symptoms associated with AD involve cognitive dysfunction, primarily memory loss (Desgranges et al., 1998; Fo¨rstl*et al.* 1995; Grafman *et al.*, 1990; Grosse *et al.* 1991). Other features associated with the later stages of AD include language deficits, depression, behavioural problems including agitation, mood disturbances and psychosis (Kumar *et al.* 1998; McGuffey, 1997; Wragg and Jeste, 1989). "Cognitive deficit" is an inclusive term used to describe the impairment of different domains of cognition.

Cognitive deficit is not limited to any particular disease or condition but may be one of the manifestations of someone's underlying condition. It is also used interchangeably with"cognitive impairment." It might be a short-term condition or a progressive and permanent entity. On the other hand, cognitive disorders are a bigger entity that is a part of neurocognitive disorders (DSM-5). Cognitive disorders are defined as any disorder that significantly impairs the cognitive functions of an individual to the point where normal functioning in society is impossible without treatment. Alzheimer disease is the most well-known condition associated with cognitive impairment. Increasing age is not only the strongest risk factor for dementia but also the only risk factor consistently identified after the eighth decade of life. Although prevalence is consistently higher among women, incidence is not; thus, the higher prevalence may largely be a function of longer life expectancy in women. Lower educational levels have been associated with higher prevalence. Within the United States, prevalence has been reported as increased in African American and Latino populations; some investigators have attributed these findings to lower education and higher cardiovascular morbidity in those populations (Julie Hugo. *et al*. 2014) Few dementias are caused by deterministic autosomal dominant genes; these are discussed later in the context of the specific disorders. Although several genes have been identified as increasing susceptibility for AD, the best-established is the apolipoprotein E (APOE) polymorphism on chromosome 19. The APOE*4 allele, associated with higher risks of hypercholesterolemia and heart disease, is also associated with dementia caused by AD, Parkinson's disease, dementia with Lewy bodies (DLB), vascular dementia, and frontotemporal dementia in men. Individuals homozygous for APOE*4 are at greater risk of dementia than those who are heterozygous. The APOE*2 seems to have a protective effect. APOE*4 is a risk factor, not a diagnostic marker for AD. It is neither necessary nor sufficient for diagnosis, and its effect on risk seems to wear off by the eighth decade (ie, individuals who are older than 80 years, APOE*4 positive, and do not yet have dementia are at no greater risk of developing dementia than those who are APOE*4 negative). (Julie Hugo. *et al*. 2014).

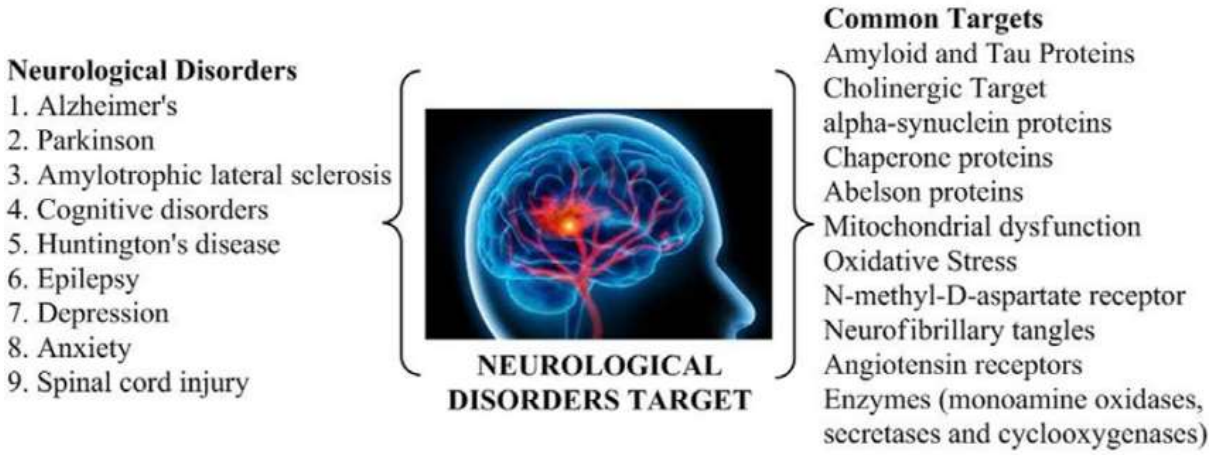
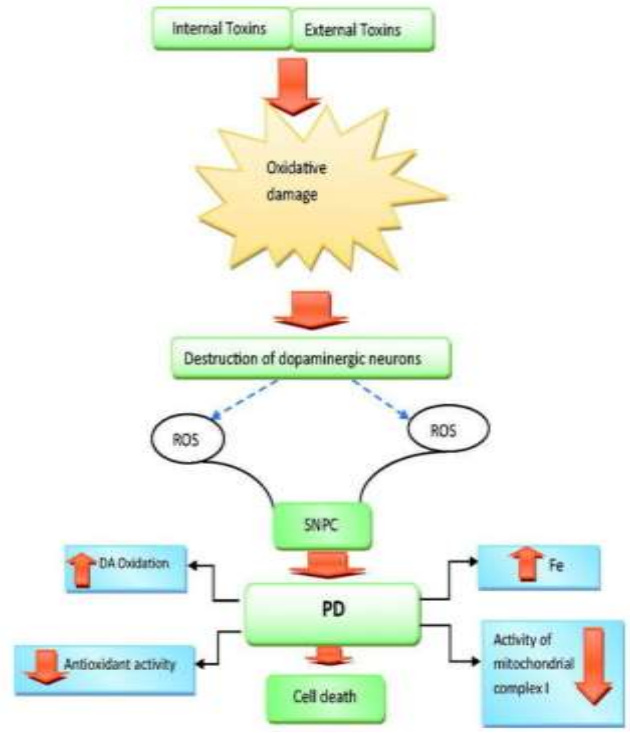


Fig 1

PARKINSON'S DISEASE

Parkinson's diseaseis a chronic midbrain *Substantia nigra* neurological disorder. Dopaminergic neuron is gradually degenerated and causes reduction of the dopaminergic level in Striatum. Tremor, dyskinesia, myotonia and so on are the signs Watched mostly in the individual suffering from the disease.



Herbal Drugs and Their Active Components with Anti-Parkinsonian Activities

The herbal medicines were listed in table 1, which, according to their families, species and part of the plant used in treatment, have been shown to be effective on PARKINSON DISEASE.

Sr. No	Plant Name	Chemical constituents	Plant Part	Uses	Remarks
1	*Acanthopanax Senticosus*	Triterpenoid Saponins, Lignans, Coumarins, and Flavones etc.	root & rhizome	Anti-Parkinson's, Anti-cancer, Anti-Stress etc	Used as adaptogen like P. ginseng
2	*Chrysanthemum indicum*	Chlorogenic acid, luteolin, and glucoside	Whole plant	Antioxidant, anti-inflammatory etc.	Scattering cold.
3	*Withaniasomnifera*	Alkaloids, anaferine, withanolides, withaferins,	Root	Anti-Stress agents. Anti Parkinson's,	"Adaptogen."
4	*Trifolium pretense*	coumestrol, phytoestrogen	Whole plant	Anti-inflammatory Muscle/ Headache	Ornamental plant.
5	*Mucuna pruriens*	Protein, Amino acid, Fatty acid, levodopa	Seed	Anti-Parkinson's, neuro-protectiveagent	Reduce Stress
6	*Bacopa monnieri*	Alkaloid brahmine, Nicotinine, herpestine.	Whole plant	Memory booster, Treated nervous disorder.	Small perennial Herbaceous plant
7	*Clausena indica*	Myristicin, terpinolene, careen etc.	Leaves	HIV infection, Anti Parkinson's drug	Antidandruff remedy
8	*Ginkgo biloba*	Flavanone, Glycoside, Biginkgosides, etc.	Whole plant	Anti-oxidant, Anti Psychiatric disorders	Treat altitude sickness (Prevention)

9	*Centella asiatica*	**Triterpenoids, brahmic acid, centellose etc.**	**Whole plant**	**Relieving anxiety, improving cognition.**	**Medicinal herb used in the orient**
10	*Ocimum sanctum*	**Oleanolic acid, ursolic acid, eugenol, carvacrol etc.**	**Whole plant**	**Antiparkinson‟s drug, Cardioprotective etc.**	**Traditional medicine**
11	*Plumbago auriculata*	**Plumbagin,isoshinanolone, plumbagin acid,vanillic acid**	**Whole plant**	**Treat a range of ailments,headache.**	**Ornamental plant**
12	*Panax ginseng*	**Ginsenosides or panaxosides, Arginine etc.**	**Whole plant**	**Anti depression agent, Anti-Parkinson's agent**	**"Adaptogen"**

Nature and Scope of The Cognitive Dysfunction Problem

The term "dementia" connotes cognitive deterioration so severe that social and occupational functioning is markedly impaired, to the extent the afflicted individual can no longer be a fully independent and productive citizen (American Psychiatricet al, 1994). As the disease progresses, personality changes emerge, and subsequently mood lability and social withdrawal take hold. Advanced dementia is characterized by progressive loss of the personality and increasing inability to perform even the simplest tasks. Mild Cognitive Impairment (MCI) features abnormal memory loss relative to one's age, but without the other changes which characterize dementia (Petersen RC et.al,1999). Age-Related Cognitive Decline (ARCD) is a diagnosis reserved for abnormal cognitive function less severe than dementia in persons older than 50. Currently, the majority of diagnosed dementias are thought to be Alzheimer's Disease, and "Alzheimer's" has now become a popular icon used to stigmatize memory problems of any degree. Sadly, this societal preoccupation with Alzheimer's is not without factual basis; within the general U.S. population one in 10 of those aged 75-85 could progress to Alzheimer's, and as many as one in three of those 85 and over. Such widespread occurrence of severe cognitive dysfunction, featuring uncoupling from one's personal history, detachment from one'ssurroundings, and finally the loss of one's very personality, is without historical precedent (Parris M.1999). A workable approach to curbing this virtual epidemic of dementia hinges on the development of a strategy for halting (or at least slowing) cognitive decline before it crosses the threshold to dementia. Research is progressing towards the definition of a "window of opportunity" for effective intervention to halt the progression to dementia. From the point of birth forward, the old adage, "Use it or lose it," applies to the brain. The brain's situation is somewhat analogous to skeletal mass, in that the more dense the brain circuitry is earlier in life, the more that can be lost later in life before function becomes seriously compromised.

Rationale for Using Indian Medicinal Plants

Table No. 2 - List plants, active compounds, and their mechanisms of action

Plant Name	**Active Compounds**	**Mechanisms**	**Evidence**
Celastrus paniculatus (Malkangni)	Celastrol, Paniculatin, β-amyrin, Sesquiterpene alkaloids	Neuroprotective, antioxidant, anti-inflammatory, nootropic	Preclinical (animal studies), limited clinical trials
Centella asiatica (Gotu Kola)	Asiaticoside	Promotes neurite outgrowth, antioxidant	Rodent models
Bacopa monniera (Brahmi)	Bacosides	Enhances synaptic transmission, antioxidant, cholinergic modulation	Human & animal studies
Withaniasomnifera (Ashwagandha)	Withanolides	Neurogenesis, GABA-mimetic, anti-inflammatory	Preclinical & clinical trials
Convolvulus pluricaulis (Shankhpushpi)	Alkaloids, flavonoids	Nootropic, anxiolytic	Animal studies
Curcuma longa (Turmeric)	Curcumin	Anti-amyloid, anti-inflammatory, antioxidant	Preclinical, limited clinical data

Neuropharmacological Investigations

Test for carbohydrates:

Presence of glycoside was determined by procedure described by:

a) Molish's test: Extract was mixed with α-naphthol solution and shaken vigorously and insoluble particles were allowed to settle. Further addition of conc.HCL was done from the edges of the test tube. Presence of violet coloured ring at the junction of mixture proved presence of carbohydrate.

b) Fehling's test: To the extract, equal quantities of Fehling's solution A and B were added and heated. Formation of a brick red precipitate indicates the presence of carbohydrates.

c) Benedict's test: 5ml of Benedict's reagent was added to extract, boiled for two minutes and cooled. Formation of a red precipitate showed the presence of carbohydrates.

Test of Tannins:

a) Ferric chloride solution was added along with extract, presence of dark blue or greenish black colour showed the presence of tannins.

b) KOH test: 1 mL of freshly prepared 10% KOH is added to 1 mL of the extract. Adirty white precipitate indicates the presence of tannins.

Test for proteins:

Biuret Test: Extract was dissolved in 1ml of 40% sodium hydroxide solution followed by addition of two drops of 1% copper sulphate solution. Formation of violet colour indicates the presence of proteins.

Test for Steroids:

Lieberman Burchardt test: Chloroform solution of the extract with few drops of acetic acid and one ml concentrated sulphuric acid gives deep red at the junction of 2 layers.

Tests for Saponins:

Foam test: A small amount of extract is shaken with little quantity of water. The foam produced persists for 10 min. It confirms the presence of saponins.

Test for Alkaloids:

a) Mayer's test (Potassium Mercuric Iodide): Extract was added with few drops of Mayer's reagent. Creamy white precipitate shows presence of alkaloid.

b) Wagner's Tests (Solution of Iodine in Potassium Iodide): Extract was added with few drops of Wagner's reagent. Presence of reddish brown coloured precipitate shows presence of alkaloids

c) Hager's Test (Saturated solution of picric acid): Extract was added with Hager's reagent. Presence of yellow precipitate shows proved presence of alkaloids.

Test for phenolic compound:

a) 5% Fecl3 solution: Deep black blue colourappear.

b) Lead acetate solution: White precipitate.

c) Gelatine solution: White precipitate.

d) Bromine water: De-coloration of bromine water.

e) Dilute potassium permanganate test: De-coloration of precipitate.

f) Potassium dichromate: red precipitate.

2. REVIEW OF LITERATURE

Singh Swetza and Gupta Rajiv (2024) highlighted the surge in mental health disorders during the post-COVID-19 era and the rising relevance of Indian medicinal plants in combating stress, anxiety, and cognitive decline. They emphasized that *Bacopa monnieri*, *Withaniasomnifera*, and *Convolvulus pluricaulis* exhibit neuroprotective and anxiolytic effects, making them valuable agents in integrative neuropsychiatric management.

Negin Holland et al. (2021) discussed the vital but often overlooked role of the noradrenergic system in cognitive processes. Their findings suggested that degeneration of the locus coeruleus, the primary source of noradrenaline, could precede other neurodegenerative changes in Alzheimer's and Parkinson's diseases, supporting early intervention strategies that include herbal nootropics.

Vijh et al. (2022) employed a network pharmacology and bioinformatics approach to explore the action of curcumin in Alzheimer's disease. Their study identified five key target genes (RARA, APP, PRARG, STAT3, and MAPK1) that are modulated by curcumin, and molecular docking revealed strong binding affinities with crucial proteins involved in neurodegeneration. This supports curcumin's multi-targeted therapeutic role and validates its use in developing tau- and amyloid-directed therapies.

Sharma Deeksha et al. (2022) reviewed traditional Ayurvedic herbs such as *Shankhpushpi* and *Ashwagandha*, noting their nootropic, anxiolytic, and antioxidant actions. These herbs, rich in flavonoids and alkaloids, modulate neurotransmitter pathways and support neurogenesis, offering therapeutic potential against age-related cognitive impairments.

Dubey et al. (2019) demonstrated that *Bacopa monnieri* extract downregulated P-glycoprotein and CYP3A enzymes in the liver and intestines. This pharmacokinetic modulation implies a strong interaction potential when co-administered with conventional drugs, which is critical for developing safe combination therapies for Alzheimer's disease.

Tewari Devesh et al. (2018) explored ethnopharmacological approaches to dementia and emphasized the neuroprotective potential of *Curcuma longa* and *Ginkgo biloba*. These botanicals attenuate β-amyloid plaque formation and oxidative stress, supporting their integration into multi-target treatment frameworks.

Aminzadeh et al. (2018) highlighted the crucial relationship between mitochondrial dysfunction, calcium imbalance, and reactive oxygen species (ROS) generation in early neurodegenerative processes. They showed that natural compounds like curcumin can restore mitochondrial homeostasis, reducing oxidative damage and improving neuronal survival. Their findings provide biochemical justification for targeting mitochondria using plant-derived antioxidants in Alzheimer's disease.

Limpeanchob et al. (2008) found that *Bacopa monnieri* extract reduced intracellular reactive oxygen species (ROS) in neuron cultures. This oxidative stress mitigation provides mechanistic evidence for its traditional use as a memory enhancer and cognitive tonic in Indian medicine.

Kumar and Gupta (2002a) investigated the cognitive effects of *Celastrus paniculatus* in rat models. Their study confirmed that seed oil exerted antioxidant effects in the CNS and improved memory through modulation of biogenic amines, suggesting its relevance in neurodegenerative conditions.

Kumar and Gupta (2002b) also demonstrated that *Centella asiatica* exerted tranquilizing and cognition-enhancing effects in animal models. These outcomes were attributed to its antioxidant properties and influence on cholinergic systems, supporting its traditional use in treating anxiety and memory loss.

Nalini et al. (1995) evaluated *Celastrus paniculatus* seed oil and found it decreased brain levels of serotonin, dopamine, and norepinephrine. The oil reversed scopolamine-induced cognitive deficits and improved learning and memory without exhibiting neurotoxicity.

Gattu et al. (1997) further confirmed the cognitive-enhancing effects of *C. paniculatus* seed oil. While not associated with cholinesterase inhibition, the oil reversed memory deficits, suggesting alternative mechanisms such as neurochemical modulation and improved synaptic function.

Schliebs et al. (1997) reported that *Withaniasomnifera* extract increased acetylcholine content and choline acetyltransferase activity in rats. These changes directly correlated with improved memory and cognitive function, confirming Ashwagandha's potential role in treating Alzheimer's-related memory loss.

Parris M. Kidd (1999) proposed an integrative model for cognitive dysfunction management, advocating the use of botanical agents like *Bacopa monnieri* and *Withaniasomnifera* for their antioxidant, anti-inflammatory, and neuroprotective effects, which align with modern neurodegenerative pathology.

Francis et al. (1999) introduced the cholinergic hypothesis, suggesting that the loss of acetylcholine is a central feature of Alzheimer's disease. This hypothesis laid the foundation for developing cholinesterase inhibitors and using cholinergic-modulating herbs like *Bacopa monnieri* and *Centella asiatica*.

Petersen et al. (1999) developed the concept of Mild Cognitive Impairment (MCI), a precursor to Alzheimer's disease, emphasizing the importance of early intervention. Herbal nootropics may be particularly effective during this "window of opportunity" before irreversible dementia sets in.

Dev S. (1997) explored the drug development potential of Ayurvedic medicines. He recommended re-examining traditional cognition-enhancing herbs like *Celastrus paniculatus* using modern scientific methods to discover active constituents and validate therapeutic claims.

3. PLANT PROFILE

Botanical Name: Bacopa monnieri.

Family: Plantaginaceae (previously classified under Scrophulariaceae)

Common Names: • English: Water hyssop, Herb of grace

• Hindi/Sanskrit: Brahmi

• Tamil: Neer Brahmi

• Malayalam: Brahmi

• Telugu: Saraswati Aku

• Kannada: Ondelaga

Plant Type: Perennial, creeping herb; semi-aquatic or aquatic plant

Morphology:

• Stem: Soft, succulent, and creeping

• Leaves: Opposite, oblong, thick, and fleshy with no prominent petiole

• Flowers: Small, actinomorphic, pale blue to white with 5 petals

• Roots: Fibrous, arising at nodes

Habitat & Distribution:

Commonly found in marshy areas, wetlands, and along riverbanks.Widely distributed throughout India, Nepal, Sri Lanka, China,Pakistan, and tropical regions of Asia, Africa, and the Americas.

Propagation:

Mainly through stem cuttings and vegetative parts; also by seeds under controlled conditions.

Phytochemical Constituents: • Major: Bacosides A & B (triterpenoid saponins)

• Others: Alkaloids (brahmine, herpestine), flavonoids, sterols, betulic acid, and otherglycosides

Medicinal Uses (Traditional & Modern):

• Cognitive enhancer: Improves memory, learning, and concentration

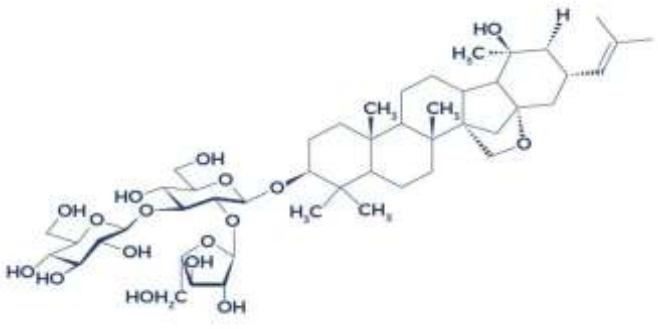
• Neuroprotective: Used in anxiety, epilepsy, and Alzheimer's disease

• Anti-inflammatory & antioxidant

• Adaptogenic and hepatoprotective properties

• Used in Ayurveda, Unani, and other traditional systems of medicine.

Chemical Structure



Bacopa monnieri has many pharmacological actions, including antioxidant, anti-inflammatory, anticonvulsant, cardiotonic, bronchodilator, and peptic ulcer protection.cognitive improvement includes modulation of acetylcholine release, muscarinic cholinergic receptor binding, and choline acetylase activity. The saponins in *Bacopa* modulate hypothalamic-pituitary-adrenal axis output and protect the hippocampus. *Bacopa* causes an anti-inflammatory effect on activated microglial cell cultures. The microglial cells respond to any injury by transforming into a neuroprotective or neurotoxic phenotype that releases pro-inflammatory cytokines.It is native to India, Indochina, Australia, and Sri Lanka. The leaves are used medicinally and contain triterpenoid saponins (Bacoside A, bacoside B, bacopasaponins, D-mannitol, acid A, monnieri), alkaloids (brahmine, nicotine, herpestine, hydrocotyline), flavonoids (luteolin, apigenin), glycosides (asiaticoside, thanakunicide), Phytochemicals (betulinic acid, betulic acid, wogonin, oroxindin, stigmasterol, beta-

sitosterol), sapogenin (jujubacogenin, pseudojujubacogenin) and other compounds (Brahmic acid, brahamoside, brahminoside, isobrahmic acid.)The saponins are believed to be responsible for most of the pharmacological actions.

4. MATERIAL AND METHODS

Material

The whole plant of *Bacopa monnieri* was collected from a local herbal nursery and authenticated by a taxonomist at Oriental College of Pharmacy, Bhopal M.P.

Methods

Preparation of Plant Material

The collected plant material was washed thoroughly with running tap water to remove dirt and adhered debris, then shade dried at room temperature (25 ± 2°C) for 7–10 days. The dried plant was coarsely powdered using a mechanical grinder and stored in an airtight container for further analysis.

Pharmacognostical Evaluation

Macroscopic Evaluation

The fresh whole plant was examined for external characteristics such as:

- Leaf shape, size, colour, and margin
- Stem structure, branching pattern
- Flower appearance
- Root morphology

Microscopic Evaluation

Microscopic examination was conducted on transverse sections (T.S.) of the stem, root, and leaf to identify diagnostic features. The sections were stained using phloroglucinol and hydrochloric acid for lignin detection and iodine for starch grains.

Table 1: Diagnostic Microscopic Features of *Bacopa monnieri*

Plant Part	**Diagnostic Feature**	**Observation**
Leaf	Epidermis with multicellular glandular trichomes	Present
Stem	Collenchymatous hypodermis, vascular bundles	Present
Root	Central stele, xylem vessels	Present
Parenchyma	Intercellular spaces with oil globules	Abundant

Physicochemical Parameters

Table 2: Physicochemical Parameters of *Bacopa monnieri* Powder

Parameter	**Observed Value (%)**
Loss on drying at 105°C	6.2 ± 0.3
Total ash	12.5 ± 0.4
Acid-insoluble ash	2.3 ± 0.2
Water-soluble ash	5.6 ± 0.3
Extractive value (water)	18.4 ± 0.5
Extractive value (ethanol)	15.1 ± 0.6
Foreign organic matter	Not more than 1%

Phytochemical Screening

The powdered drug was subjected to preliminary phytochemical tests to detect the presence of major constituents.

Table 3: Preliminary Phytochemical Screening of *Bacopa monnieri*

Phytochemical Group	Test Performed	Result
Alkaloids	Dragendorff's and Mayer's	+ve
Glycosides	Keller–Killiani test	+ve
Saponins	Froth test	+ve
Flavonoids	Shinoda test	+ve
Tannins	Ferric chloride test	+ve
Proteins	Biuret test	-ve
Steroids	Liebermann–Burchard test	+ve

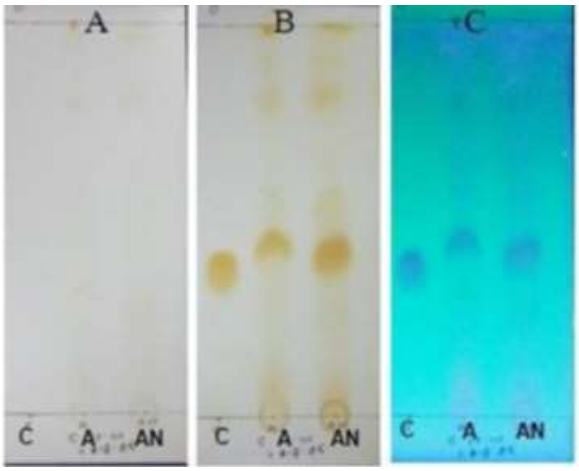
Extraction Procedure

Approximately 100 g of powdered *Bacopa monnieri* was extracted with 500 mL of methanol using a Soxhlet extractor for 6–8 hours. The extract was filtered, concentrated using a rotary evaporator, and stored at 4°C for further pharmacological evaluation.

Chromatographic Fingerprinting

TLC (Thin Layer Chromatography)

- Solvent system: Chloroform:Methanol:Water (65:25:4)
- Detection: Iodine vapours and UV at 254 & 366 nm
- Marker compound: Bacoside A (Rf ~0.54)



Fluorescence Analysis

Powdered drug was treated with various reagents and observed under UV light (254 nm and 366 nm) and visible light. Table 4

Reagent	Daylight Colour	UV 254 nm	UV 366 nm
Powder alone	Greenish brown	Dark green	Pale green
1N NaOH (aq)	Brown	Fluorescent green	Yellowish green
1N HCl	Brownish pink	Pale orange	Greenish yellow
Methanol	Greenish brown	Light green	Greenish brown

5. RESULT AND DISCUSSION

Macroscopic inspection confirmed the typical soft, creeping stems, opposite fleshy leaves and pale-blue corolla of *Bacopa monnieri*. Microscopy showed multicellular glandular trichomes on the epidermis, a collenchymatous hypodermis with collateral vascular bundles in the stem, and a di-arch stele in the root—features that match the Ayurvedic Pharmacopoeia monograph, confirming botanical identity.

Physicochemical parameters(mean ± SD, n = 3)

Loss on drying (LOD) = **6.2 ± 0.3 % w/w**

Total ash = **12.5 ± 0.4 % w/w**

Acid-insoluble ash = **2.3 ± 0.2 % w/w**

Water-soluble ash = **5.6 ± 0.3 % w/w**

Water-soluble extractive = **18.4 ± 0.5 % w/w**

Alcohol-soluble extractive = **15.1 ± 0.6 % w/w**

Foreign organic matter < 1 %

Preliminary phytochemical screening

Alkaloids, glycosides, saponins, flavonoids, tannins and steroids were all positive, whereas proteins were absent.

Chromatographic fingerprinting

TLC on silica gel with $CHCl_3$:MeOH:H_2O (65:25:4) produced a major band at Rf ≈ 0.54, identical to the bacoside-A reference, and a characteristic poly-spot profile under iodine vapour, establishing a reproducible chemical fingerprint.

Fluorescence analysis

Powder exhibited greenish fluorescence in daylight that intensified to bright yellow-green with 1 N NaOH under 366 nm UV, providing a rapid identity test for crude material.

Discussion

Compliance with Pharmacopeial limits

The Ayurvedic Pharmacopoeia (API, Part I Vol II) prescribes LOD ≤ 7 % and total ash ≤ 7 % for whole-plant *B. monnieri* , while several contemporary quality-control studies set broader criteria (e.g., LOD ≤ 10 %, total ash ≤ 13 %) for aerial parts dried under field conditions

- The present LOD (6.2 %) is just above the Indian Pharmacopoeia cap of 6 % for dry herbal drugs but still within the wider WHO/Research limits, suggesting only marginally elevated residual moisture—probably a consequence of shade-drying in a humid climate. Air-oven finish-drying (48 h, 40 °C) or freeze-drying can safely reduce residual water without bacoside loss.
- Total ash at 12.5 % exceeds the API value but respects the 13 % ceiling quoted for aerial parts in recent stability studies. The higher mineral residue is often linked to swampy collection sites and adventitious sand; meticulous washing and a final tap-water rinse followed by de-ionised water can reduce inorganic contamination.
- Acid-insoluble ash (2.3 %) marginally exceeds both API (≤1 %) and WHO (≤2 %) thresholds, again pointing to siliceous matter. Future batches should incorporate a flotation or sieving step before drying.

Phytochemical profile and neuropharmacological relevance Strong reactions for saponins (bacosides), flavonoids and alkaloids corroborate the nootropic and neuroprotective claims widely reported for *B. monnieri*. Bacosides scavenge ROS, up-regulate BDNF and modulate cholinergic signalling, aligning with the study's broader aim of integrative management of cognitive disorders. Positive flavonoid and tannin tests add anti-inflammatory synergy, while steroidal constituents may contribute adaptogenic effects.

TLC and fluorescence as quality-control markersThe single dominant Rf 0.54 bacoside-band observed here overlaps the bacoside-A zone cited in the API monograph, giving a simple yet definitive identity and purity check. Combined with the diagnostic fluorescence matrix, these low-cost assays provide a rugged in-house QC package for routine batch release.

Limitations and future work Quantitative HPLC (e.g., bacoside-A ≥ 3.5 % w/w) was not performed in this pilot; incorporating it would allow direct comparison with pharmacodynamic endpoints. In vivo antioxidant and cholinesterase assays on the present extract will help correlate the rich phytochemistry with functional neuroprotection.

6. CONCLUSION

The present investigation supports the traditional use of *Bacopa monnieri* as a neuroprotective agent in the management of cognitive disorders. The pharmacognostical and phytochemical assessments confirm the identity, purity, and richness of bioactive constituents relevant to cognitive health, particularly bacosides, alkaloids, and flavonoids. TLC fingerprinting and fluorescence analysis provide reliable tools for quality control and standardization of herbal formulations. While the findings align with previous literature on the plant's nootropic potential, future studies should

incorporate advanced analytical techniques like HPLC and in vivo neuropharmacological assays to validate and quantify its therapeutic efficacy. Integrating *Bacopa monnieri* into modern neurotherapeutic regimens may offer a safe, effective, and holistic approach to addressing age-related cognitive decline and neurodegenerative conditions.

7. BIBLIOGRAPHY

Abdul, M.; Aimi, S.; *et al.*2019 "Bacopa monnieri, a Neuroprotective Lead in Alzheimer Disease: A Review on Its Properties, Mechanisms of Action, and Preclinical and Clinical Studies." *Drug target insights, 13*,

Ahmad F, Khan RA, Rasheed S. 1994 “Preliminary screening of methanolic extracts of Celastrus paniculatus and Tecomella undulata for analgesic and anti-inflammatory activities” J Ethnopharmacol ;42:193 – 8

Asakawa Y, Matsuda R, Takemoto T. 1982 “Monoterpenoids and sesquiterpenoids from Hydrocotyle and Centella species” Phytochemistry;21(10): 2590 – 2.

American Psychiatric Association1994 “Diagnostic and Statistical Manual of Mental Disorders”.Washington, DC: American Psychiatric Association.

Angelucci, F., Cechova, K., Valis, M., Kuca, K., Zhang, B., and Hort, J.2019“MicroRNAs in alzheimer’s disease: Diagnostic markers or therapeutic agents? Front” Pharmacol. 10, 665.

Aguiar, S.; Borowski, T. 2013 “Neuropharmacological review of the nootropic herb Bacopa monnieri” RejuvenationRes, *16*, 313-326,

Banasch, M., Ellrichmann, M., Tannapfel, A., Schmidt, W. E., and Goetze, O. 2011 “The non-invasive (13)C-methionine breath test detects hepatic mitochondrial dysfunction as a marker of disease activity in non-alcoholic steatohepatitis” *Eur. J. Med. Res.* 16:258.

Beal, M. F. 2005 “Mitochondria take center stage in aging and neurodegeneration” *Ann. Neurol.* 58, 495–505.

Bidwai PP, Wangoo D, Bhullar NK. 1987 “Effect of Celastrus paniculatus seed extract on the brain of albino rats” J Ethnopharmacol;21: 307 – 14.

Brinkhaus B, Lindner M, Schuppan D, Hahn EG. 2000 “Chemical, pharmacological and clinical profile of the East Asian medicinal plant Centella asiatica” Phytomedicine;7(5):427 – 48

Burte, F., Carelli, V., Chinnery, P. F., and Yu-Wai-Man, P. 2015 “Disturbed mitochondrial dynamics and neurodegenerative disorders” *Nat. Rev. Neurol.* 11, 11–24.

Chainoglou, E., and Hadjipavlou-Litina,D. 2020 “Curcumin in health and diseases: Alzheimer’s disease and curcumin analogues, derivatives, and hybrids” Int. J. Mol. Sci. 21, 1975.

Chen, F., Eckman, E. A., and Eckman, C. B. 2006 “Reductions in levels of the Alzheimer’s amyloid beta peptide after oral administration of ginsenosides” FASEB J. Off. Publ. Fed. Am. Soc. Exp. Biol. 20, 1269–1271

Costa, A. C. D. S., Menon, V., Phadke, R., Dapke, K., Miranda, A. V., Ahmad, S., & Hashim, H. T. 2022 “Mental health in the post COVID-19 era: futureperspectives”*Einstein (São Paulo), 20*

Crook TH, Adderly B.*The Memory Cure*. NewYork, NY: Simon and Shuster; 1998.

Desgranges B, Baron J-C, de la Sayette V, Petit-Taboue´ M-C, Benali K, Landeau B*et al.* 1998 “The neural substrates of memory systems impairment in Alzheimer’s disease” Brain; 121:611 – 31.

Dev S. 1997 “Ethnotherapeutics and modern drug development: the potential of Ayurveda” Curr Sci;73(11):909 – 28.

Duan, Q., Jing, Z., Zou, X., Wang, Y., Yang, K., Zhang, T.,*et al*. 2020 “Spiking neurons with spatiotemporal dynamics and gain modulation for monolithically integrated memristive neural networks” Nat. Commun. 11, 1–13.

Dubey, T.; Chinnathambi, S. 2019 “Brahmi (Bacopa monnieri): An ayurvedic herb against the Alzheimer's disease” Arch. Biochem. Biophys., *676*,

Duke JA, Ayensu ES. 1985 Medicinal plants of China. Vols. 1 – 2. 2nd ed. Algonac (MI): Reference Publications.

Fields, C., Bischof, J., and Levin, M. 2020 “Morphological coordination: A common ancestral function unifying neural and non-neural signaling” Physiology 35, 16–30.

Fo¨ rstl H, Hentschel F, Sattel H, Geiger-Kabisch C, Besthorn C, Czech C*et al.* 1995 “Age-associated memory impairment and early Alzheimer’ disease” Drug Res;45(1):394 – 7

Francis PT, Palmer AM, Snape M, Wilcock GK. *et al.* 1999 “The cholinergic hypothesis of Alzheimer’s disease” a review of progress. J NeurolNeurosurg Psychiatry;66(2):137 – 47

Gattu M, Boss KL, Terry AV, Buccafusco JJ.*et al.* 1997 “Reversal of scopolamine-induced deficits in navigational memory performance by the seed oil of Celastrus paniculatus” PharmacolBiochem Behav;57(4): 793 – 9.

Grafman J, Weingartner H, Lawlor B, Mellow AM, Thompsen-Putnam K, Sunderland T.*et al.* 1990 Automatic memory processes in patients with dementia—Alzheimer's type (DAT). Cortex; 26:361 – 71.

Greenberg, N., Brooks, S. K., Wessely, S., & Tracy, D. K. 2020 "How might the NHS protect the mental health of health-care workers after the COVID-19 crisis?"*The Lancet Psychiatry*, *7*(9), 733-734.

Grosse DA, Gilley DW, Wilson RS. 1991 "Episodic and semantic memory in early versus late onset Alzheimer's disease" Brain Lang; 41:531 – 7.

Hewlings, S. J., and Kalman, D. S. 2017. "Curcumin: A review of its effects on human health. Foods" (Basel, Switz. 6, 92.

Holcomb, L.A.; Dhanasekaran, M.; Hitt, R.; Young, K.A.; Riggs, M.; Manyam, B.V. 2006 "Bacopa monnieraextract reduces amyloid levels in PSAPP mice"*J Alzheimers Dis*., *9*, 243–251.

Janas, A. M.; Cunningham, S. C.; Duffy, K. B.; Devan, B. D.; Greig, N. H.;Holloway, H. W.; Yu, Q.-S.; Markowska, A. L.; Ingram, D. K.; Spangler, E. L. 2005 "The Cholinesterase Inhibitor, Phenserine, Improves Morris Water Maze Performance of Scopolamine-Treated Rats. *Life Sci*., *76* (10), 1073–1081.

Jeyasri R, Muthuramalingam P, Suba V, Ramesh M, Chen JT. 2020 "*Bacopa monnieri* and Their Bioactive Compounds Inferred Multi-Target Treatment Strategy for Neurological Diseases: A Cheminformatics and System Pharmacology Approach. Biomolecules" Apr 2;10(4):536.

Jensen, N. J., Wodschow, H. Z., Nilsson, M., and Rungby, J. 2020 "Effects of ketone bodies on brain metabolism and function in neurodegenerative diseases" Int. J. Mol. Sci. 21, 8767.

Julie Hugo, MD, Mary Ganguli, MD, MPH*et al.* 2014 "Dementia and Cognitive Impairment" Clinics in Geriatric Medicine; 30:421-442.

Jyotirmayee, B., and Mahalik, G. 2022 "A review on selected pharmacological activities of Curcuma longa" L. Int. J. Food Prop. 25, 1377–1398.

Kapoor LD. 1990 Handbook of Ayurvedic medicinal plants. Boca Raton (FL): CRC Press.

Khyati, Malik, I., Agrawal, N., and Kumar, V. 2021 "Melatonin and curcumin reestablish disturbed circadian gene expressions and restore locomotion ability and eclosionbehavior in Drosophila model of Huntington's disease"Chronobiol. Int. 38, 61–78.

Kumar, A.; Singh, A. 2015 **"**A Review on Alzheimer's Disease Pathophysiology and ItsManagement: An Update"*Pharmacol. Reports*, *67* (2), 195–203

Kumar MHV, Gupta YK. 2002 "Antioxidant property of Celastrus paniculatus Willd.: a possible mechanism in enhancing cognition" Phytomedicine;9(4):302 – 11.

Kumar, S.; Christopher, J.S.; Edward, J.O. 2011 "Kinetics of acetylcholinesterase inhibition by an aqueous extractof Withaniasomnifera roots"*Int. J. Pharm. Sci. Res.*, *2*, 1188-92

Kumar V, Durai NB, Jobe T. 1998 "Pharmacologic management of Alzheimer's disease" Clin Geriatr Med;14(1):129 – 46.

Kumar, V.2006 "Potential medicinal plants for CNS disorders: an overview"*Phytother Res*, *20*, 1023-1035

Limpeanchob, N.; Jaipan, S.; Rattana, karuna, S.*et al*.2008"Neuroprotective effect of Bacopa monnieri on beta–amyloid–induced cell death in primary cortical culture"*J Ethnopharmacol.*, *120*, 112–127

Lopresti, A. L. 2018 "The problem of curcumin and its bioavailability: Could its gastrointestinal influence contribute to its overall health-enhancing effects?" Adv. Nutr. 9, 41–50.

Manyam BV. 1999 "Dementia in Ayurveda". J Altern Complement Med; 5(1):81 – 8.

McGuffey EC. 1997 Alzheimer's disease: an overview for the pharmacist. JAMA; NS37(3):347 – 52.

Melanie-Jayne R. Howesa, Peter J. Houghton 2003 "Plants used in Chinese and Indian traditional medicine for improvement of memory and cognitive function" Pharmacology, Biochemistry, and Behaviour;75:513-527

Miyazawa M, Watanabe H, Kameoka H. 1997 "Inhibition of acetylcholinesterase activity by monoterpenoids with a p-menthane skeleton" J Agric Food Chem; 45:677 – 9.

Moreira, P. I., Carvalho, C., Zhu, X., Smith, M. A., and Perry, G. 2010 "Mitochondrial dysfunction is a trigger of Alzheimer's disease pathophysiology" *Biochim. Biophys. Acta* 1802, 2–10.

Mukherjee, S.; Dugad, S.; Bhandare, R.;*et al*.2011 "Evaluation of comparative free-radical quenching potential ofBrahmi (Bacopa monnieri) and Mandookparni (Centella asiatica)"*Ayu*., *32*, 258–264,

Nadkarni KM. 1976 **"**Indian materia medica". 3rd ed. Bombay: Popular Prakashan.

Nalini K, Karanth KS, Rao A, Aroot AR. 1995 "Effects of Celastrus paniculatus on passive avoidance performance and biogenic amine turnover in albino rats" J Ethnopharmacol;47:101 – 8.

Negin Holland, Trevor W. Robbins, and James B. Rowe*et al.* 2021 "The role of noradrenaline in cognition and cognitive disorders" Brain Review Article, 144;2243-2256.

Parris M. Kidd, PhD 1999 "A Review of Nutrients and Botanicals in the Integrative Management of Cognitive Dysfunction" Alternative Medicine Review; (4):144-161

Pathophysiology of Alzheimer's disease - Oxford Medicine9780199569854-chapter-4(accessed May 23, 2016)

Perry NSL, Houghton PJ, Theobald A, Jenner P, Perry EK. 2000a "In-vitro inhibition of human erythrocyte acetylcholinesterase by Salvia lavandulaefolia essential oil and constituent terpenes" J Pharm Pharmacol; 52:895 – 902.

Petersen RC, Smith GE, Waring SC, et al. 1999 "Mild cognitive impairment: clinical characterization and outcome"*Arch Neurol*;56:303-308.

Russo, A.; Borrelli, F.2005 "Bacopa monniera, a reputed nootropic plant: an overview" Phytomedicine, 12,305-317,

Russo, A.; Izzo, A.A.; Cardile, V.;*et al.* 2001 **"**Indian medicinal plants as antiradicals and DNA cleavage protectors.Phytomedicine., *8*, 125–132,

Ryan MF, Byrne O. 1988 "Plant-insect coevolution and inhibition of acetylcholinesterase" J Chem Ecol;14(10):1965 – 75

Sandhu, J.S.; Shah, B.; Shenoy, S.; Chauhan, S.; Lavekar, G.S.; Padhi, M.M.2010 "Effects of Withaniasomnifera(Ashwagandha) and Terminalia arjuna (Arjuna) on physical performance and cardiorespiratory endurance inhealthy young adults" Int J Ayurveda Res., *1*, 144-9,

Sakina MR, Dandiya PC. 1990 "A psycho-neuropharmacological profile of Centella asiatica extract" Fitoterapia;61(4):291 – 6.

Saraf, M.K.; Prabhakar, S.; Khanduja, K.L.; Anand, A. 2011 "Bacopa monniera attenuates scopolamine-induced impairment of spatial memory in mice. *Evid Based Complement Alternat Med*.

Sama, D. M., and Norris, C. M. 2013 "Calcium dysregulation and neuroinflammation: discrete and integrated mechanisms for age-related synaptic dysfunction" *Ageing Res. Rev.* 12, 982–995.

Schliebs, R.; Liebmann, A.; Bhattacharya, S.K.;*et al*. 1997 **"**Systemic administration of defi ned extracts fromWithaniasomnifera (Indian Ginseng) and Shilajit differentially affects cholinergic but not glutamatergic andGABAergic markers in rat brain". *Neurochem Int*., *30*, 181–190.

SharmaDeeksha,TyagiSakshi, DanSiddhartha*et al.* 2022 "Indigenous Therapeutics of Alzheimer: A Review of Ayurvedic Herbs from its Ethnobotany to Phytotherapy" Platinum Open Access Journal; 11(4):4178-4191.

Shinomol, G.K.; Muralidhara, Bharat. M.M. 2011 "Exploring the role of 'Brahmi' (Bacopa monnieri and Centella asiatica) in brain function and therapy"*Recent Pat EndocrMetab Immune Drug Discov.*, *5*, 33–49

Singh, R.; Panduri, J.; Kumar, D.;*et al*. 2013 **"**Evaluation of memory enhancing clinically available standardizedextract of Bacopa monniera on P-glycoprotein and cytochrome P450 3A in Sprague-Dawley rats. *PLoS ONE*., *8*

Singh, S. 2012 **"**Phytochemical analysis of leaf callus of Bacopa monnieri" L. Int J Sci Res Pub, 2, 1-3

SinghSwetzaand Gupta Rajiv 2024 "Ethno-pharmacological Activities of Some Important Medicinal Plants on Mental Health" J. Pharm. Tech. Res. Management 12:1-13.

Sivanantharajah, L., and Mudher, A. 2022 "Curcumin as a holistic treatment for tau pathology" Front. Pharmacol. 13, 903119.

Swerdlow, R. H., Burns, J. M., and Khan, S. M. 2010 "The Alzheimer's disease mitochondrial cascade hypothesis" *J. Alzheimers. Dis.* 20(Suppl. 2), S265–S279.

Tewari Devesh, Stankiewicz Adrian M. *et al.* 2018"Ethnopharmacological Approaches for Dementia Therapy and Significance of Natural Products and Herbal Drugs" Frontiers in Aging Neuroscience; 10 (3): 1-24

van Horssen, J., van Schaik, P., and Witte, M. 2017 "Inflammation and mitochondrial dysfunction: a vicious circle in neurodegenerative disorders?" Neurosci. Lett.

Vijh, D., Imam, M., Haque, M., Das, S., Islam, A., and Malik, M. 2022 "Network pharmacology and bioinformatics approach reveals the therapeutic mechanism of action of curcumin in Alzheimer disease. Res. Sq. Prepr. version. 1. 1.

Watkins, P.B. 1997 **"**The barrier function of CYP3A4 and P-glycoprotein in the small bowel"*Adv Drug Deliv Rev*., *27*, 161–170,

Walsh, C., Barrow, S., Voronina, S., Chvanov, M., Petersen, O. H., and Tepikin, A. 2009 "Modulation of calcium signalling by mitochondria" *Biochim. Biophys. Acta* 1787, 1374–1382.

Wang, C., and Youle, R. J. 2009 "The Role of Mitochondria in Apoptosis" *Annu. Rev. Genet.* 43, 95–118.

Warrier PK, Nambiar VPK, Ramankutty C. 1995 Indian medicinal plants, vol. 2. India: Orient Longman.

Wragg RE, Jeste DV. 1989 "Overview of depression and psychosis in Alzheimer's disease" Am J Psychiatry; 146:577 – 87.

Yang, J. Q., Wang, R., Ren, Y., Mao, J. Y., Wang, Z. P., Zhou, Y.,*et al*. 2020 "Neuromorphic engineering: From biological to spike-based hardware nervous systems" Adv. Mat. 32, 2003610.

Zhu, X., Perry, G., Smith, M. A., and Wang, X. 2013 "Abnormal mitochondrial dynamics in the pathogenesis of Alzheimer's disease" *J. Alzheimers. Dis.* 33(Suppl. 1), S253–S262.