PHARMA RESEARCH BULLETIN
Open Access Journal (ISSN: 2582-676X)

Review article
Volume 5, Issue 1, 2026, 109-123

Brahmi and Its Extracts in the Administration of Neurodegenerative Syndromes

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Received: 15 October 2025; Accepted: 05 January 2026; Published: 19 January 2026

Abstract: Neurodegenerative syndromes are progressive disorders characterized by the irreversible loss of neuronal structure and function, leading to cognitive decline, motor impairment, and reduced quality of life. Despite advances in pharmacotherapy, current treatment strategies remain largely symptomatic and are often associated with adverse effects, necessitating the exploration of safer and more effective alternatives. *Bacopa monnieri* (Brahmi), a well-known medicinal herb in traditional systems of medicine, has gained increasing scientific attention due to its neuroprotective potential. This review comprehensively summarizes the pharmacological properties of *B. monnieri*, with particular emphasis on its bioactive constituents, including bacosides, and their mechanisms of action in neurodegenerative syndromes. Experimental and clinical studies suggest that Brahmi exerts antioxidant, anti-inflammatory, cholinergic-modulating, and neuroprotective effects, contributing to improvements in memory, cognition, and neuronal survival. The evidence presented highlights the therapeutic relevance of *B. monnieri* as a promising candidate for the management of neurodegenerative disorders and supports further research aimed at its clinical translation and formulation development.

Keywords: Brahmi, *Bacopa monnieri*, Neurodegenerative syndromes, Bacosides, Neuroprotection, Cognitive enhancement, Memory improvement.

1. Introduction

Neurodegenerative diseases represent a heterogeneous group of hereditary disorders characterized by progressive and irreversible loss of neuronal structure and function. These disorders are a major cause of morbidity in the elderly population, accounting for approximately 60–80% of all mental illnesses in older individuals, with Alzheimer's disease (AD) being the most prevalent [1]. Neurodegeneration primarily affects cognition and associated functions through mechanisms such as neuronal loss, abnormal protein aggregation, oxidative stress, neuroinflammation, neurotransmitter imbalance, and impairment of endogenous neuroprotective systems [2]. Currently, an estimated 5.5 million individuals in the United States alone are affected by AD [3]. Alzheimer's disease is clinically characterized by irregular behavior, impaired thinking ability, personality alterations, and progressive cognitive dysfunction [4]. Early manifestations include short-term memory loss, mood fluctuations, difficulty in comprehension, and problems recalling conversations, recent events, and familiar names [5]. As the disease advances, more severe symptoms such as confusion, disorientation, and difficulties in eating and walking become prominent [6]. Evidence suggests that anxiety and memory impairment in AD are closely associated with degeneration of cholinergic neuron-rich regions of the brain [7]. Acetylcholine (ACh) plays a pivotal role in learning and memory processes, and dysfunction of the cholinergic system is a hallmark of AD pathology [5-7]. Accordingly, acetylcholinesterase inhibitors (AChEIs) constitute one of the most important classes of drugs approved by the US Food and Drug Administration (USFDA) for the management of AD [8]. The cholinergic system is crucial for the synchronization of learning and memory mechanisms [9]. During the early stages of amyloid plaque formation, both acetylcholinesterase (AChE) and butyrylcholinesterase (BChE) contribute to amyloid-β aggregation [10].

Inhibition of these enzymes increases acetylcholine levels in the brain, thereby reducing plaque formation. Although AChE and BChE share significant structural and functional similarities, including their roles in acetylcholine hydrolysis, inhibition of peripheral BChE can lead to serious adverse effects. Consequently, the development of selective AChE inhibitors with reduced side effects has become an area of significant research interest [11]. Currently approved AChEIs, such as donepezil, tacrine, galantamine, and rivastigmine, offer only limited therapeutic benefits. Their clinical use is often associated with cholinergic side effects, including dermatitis, hepatotoxicity, muscle cramps, nausea, fatigue, and gastrointestinal discomfort [12]. Therefore, there is a pressing need to develop more potent, effective, and safer therapeutic agents for AD management. In this context, plant-derived natural compounds have gained increasing attention due to their broad pharmacological potential and ability to address therapeutic gaps in various human disorders [13].

Herbal medicines have long been regarded as cognitive enhancers, particularly for memory and learning. Numerous medicinal plants alleviate the symptoms of Alzheimer's disease and memory impairment by modulating multiple biochemical and molecular pathways [14]. Among these, Salvia officinalis has shown promising neuroprotective effects [15]. Huperzine, isolated from Huperzia serrata, exhibits potent cholinesterase inhibitory activity and enhances acetylcholine levels [16]. Similarly, extracts of Ginkgo biloba have been reported to improve cognitive and memory performance [17].

Additionally, *Bacopa monnieri* (Brahmi) is well recognized for its cognition-enhancing properties [18]. However, a comprehensive understanding of its therapeutic relevance in neurological and memory-related disorders requires detailed knowledge of its active phytochemical constituents and underlying mechanisms of action. Antioxidant-rich extracts of *B. monnieri* protect the brain against oxidative damage and age-related cognitive decline by modulating multiple signaling pathways. The cognitive-enhancing effects of *B. monnieri* are largely attributed to bacosides, which possess strong antioxidant properties [19]. Animal studies have demonstrated that bacosides and *B. monnieri* extracts significantly enhance antioxidant status [20] and reduce diabetes-induced oxidative stress [21]. Furthermore, bacoside A has been shown to reduce free radical generation through its antioxidant activity, thereby protecting against cerebrovascular disorders induced by cigarette smoking [22]. Prolonged in vivo exposure to cigarette smoke has been reported to increase oxidative stress, further highlighting the neuroprotective potential of bacosides. Extensive research has documented the analgesic, antioxidant, antibacterial, and nootropic properties of Brahmi extract, along with its diverse bioactive constituents. Numerous experimental studies and clinical trials have validated these traditional pharmacological claims [23]. Consequently, *Bacopa monnieri* has emerged as a versatile medicinal plant with significant therapeutic potential for neurological disorders and cognitive impairments.

2. Bioactive components and their role in the body

Bacopa monnieri is rich in a wide array of physiologically important secondary metabolites, including sugars, amino acids, flavonoids, cucurbitacins, alcohols, steroids, alkaloids, glycosides, sterol glycosides, and phenylethanoid glycosides [24-26]. Several amino acids and nitrogen-containing compounds, such as brahmin, hydrocotyline, nicotine, and herpestine, have also been identified in *B. monnieri* extracts. Among these constituents, saponins constitute the major bioactive components and include betulinic acid, bacosides, bacopasides [26], and bacosaponins [27-28]. The chemical structures of these compounds are illustrated in **Figure 1**. Bacosides are the most significant phytoconstituents of *Bacopa monnieri* and play a crucial role in maintaining neuronal health. A total of twelve bacoside analogues have been isolated and structurally characterized [27, 29]. Structurally, these compounds are triterpenoid saponins in which sugar moieties are attached exclusively at the C-3 position of the aglycone, forming monodesmosides, or at both the C-3 and C-20 positions, resulting in bidesmosides [30].

Bacosides have been shown to inhibit cytotoxicity and DNA damage in Alzheimer's disease–associated neurons and to promote neuronal repair by enhancing enzymatic activity and cortical protein synthesis [31]. The majority of the neuropharmacological and nootropic activities of *B. monnieri* are attributed to bacosides A and B [29], which are widely used for memory enhancement. Bacoside A, in particular, has been reported to facilitate cerebral blood flow through nitric oxide–mediated mechanisms and to exhibit anticarcinogenic properties. In addition to bacosides, *Bacopa monnieri* contains several other bioactive compounds, including cucurbitacins, apigenin, flavonoids, and phenylethanoid glycosides [32-33]. Three phenylethanoid glycosides have been successfully isolated from the plant. Furthermore, two additional saponins and a novel glycoside, along with several other noteworthy saponins, have been identified and reported from *B. monnieri* [34].

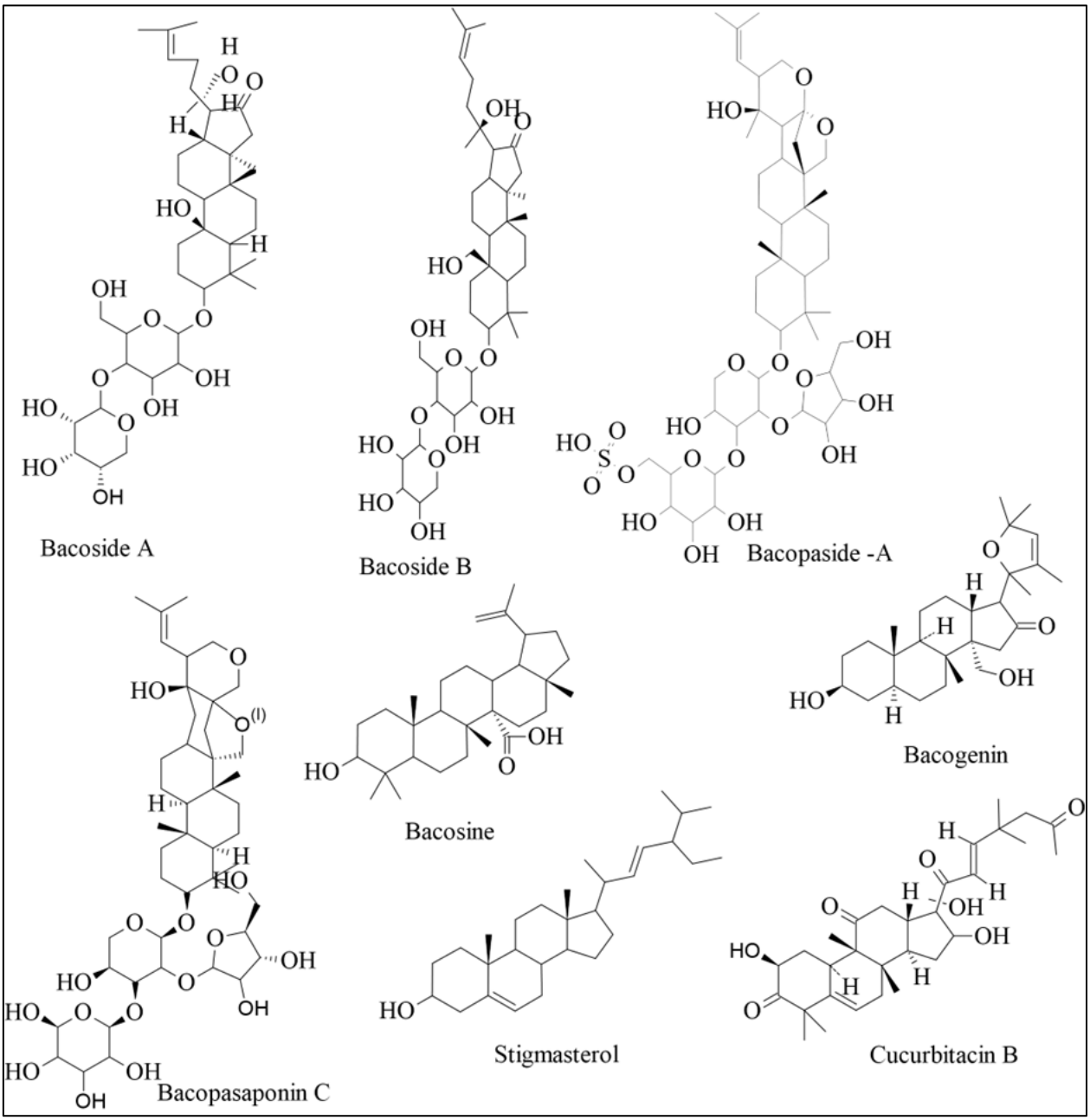

Figure 1: Structures of active constituents of *Bacopa monnieri*.

3. Neuropharmacological Action of Bacoside A

Bacosides, the principal bioactive constituents of *Bacopa monnieri*, are dammarane-type triterpenoid saponins. They exist mainly in two aglycone forms, jujubogenin and pseudojujubogenin, which differ primarily in the number and arrangement of sugar moieties within the glycosidic chain. Among these compounds, bacoside A is considered the most potent neuropharmacological agent. Bacoside A has been shown to induce membrane dephosphorylation, a process that may enhance protein and RNA synthesis in specific regions of the brain. Through these mechanisms, bacosides provide protection against cold stress–induced damage, memory impairment, oxidative stress, and age-related cognitive decline. Additionally, bacoside A reduces lipoxygenase activity, enhances cerebral blood flow, and inhibits hydrogen peroxide–induced lipid peroxidation [34-36]. These properties collectively contribute to its anti-dementia effects. Furthermore, bacoside A supports neuronal regeneration and synaptic repair by increasing protein kinase activity, particularly in the hippocampal region, thereby facilitating the restoration of damaged neurons [37-38]. Cognitive performance is also improved through modulation of serotonergic signaling, mediated by the activation of tryptophan hydroxylase-2 (TPH2) and increased expression of the serotonin transporter (SERT) [39].

4. *Bacopa monnieri's* Neuroprotective Properties

The neuroprotective effects of *Bacopa monnieri* are largely attributed to its ability to enhance endogenous antioxidant defense systems by stimulating specific enzymes and reducing oxidative stress. The plant has also been reported to decrease the accumulation of harmful metal ions, such as iron and copper, which exacerbate oxidative damage in the brain [24]. Bioactive compounds present in *B. monnieri* can modulate cholinergic neurotransmission by either activating choline acetyltransferase, the enzyme responsible for acetylcholine synthesis, or inhibiting acetylcholinesterase, the enzyme involved in acetylcholine degradation. Regulation of acetylcholine levels is crucial for cognitive function and may contribute to improved learning and memory. Additionally, *B. monnieri* has been shown to reduce β-amyloid accumulation in the brain, a hallmark pathological feature of Alzheimer's disease.

Synaptic plasticity, which plays a central role in brain function, can be rapidly modified in response to physiological and pathological stimuli [40-42]. Neurodegenerative disorders are characterized by progressive neuronal deterioration, leading to impaired motor function, cognitive decline, and overall reduction in brain health. Several disease-specific proteins have been implicated in these disorders. For instance, acetylcholinesterase (AChE) is associated with dementia and amnesia, α-synuclein with Parkinson's disease, β-amyloid with Alzheimer's disease, and vesicular glutamate receptors with schizophrenia [43-44]. Moreover, epileptic seizures have been linked to the glutamate-8 receptor (mGluR8). Understanding the molecular targets involved in neurodegenerative diseases is essential for advancing therapeutic strategies and improving patient outcomes. *Bacopa monnieri* is believed to exert neuroprotective effects by inhibiting acetylcholinesterase activity, thereby preventing the excessive breakdown of acetylcholine. This mechanism may counteract the neurotoxic effects of elevated AChE levels induced by amyloid peptides, which are commonly observed in neurodegenerative conditions. Collectively, these findings support the potential neuroprotective role of *Bacopa monnieri* in maintaining cognitive health (**Figure 2**).

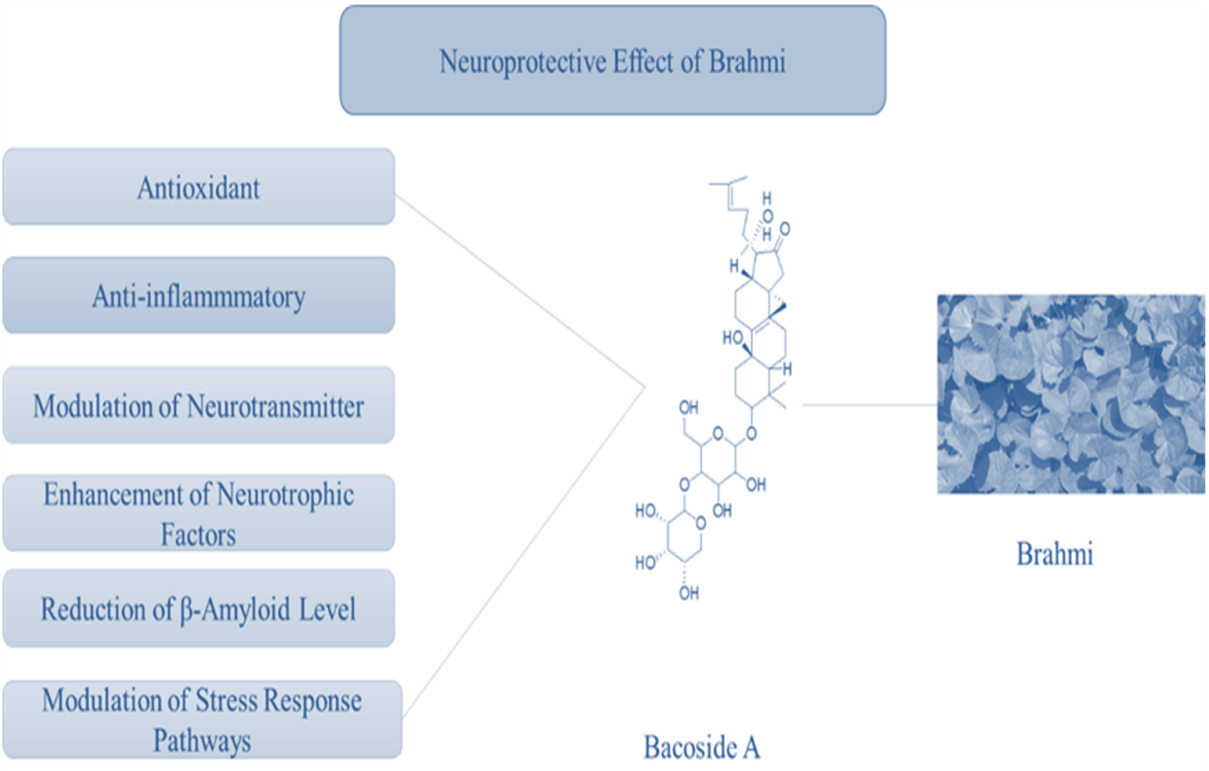

Figure 2: Neuroprotective Effect of Brahmi (*Bacopa monnieri*).

5. Pharmacological Studies of *Bacopa monnieri*

Pharmacological studies have extensively explored the therapeutic potential of *Bacopa monnieri* (Brahmi), as summarized in **Table 1**. In addition to its cognitive-enhancing effects, Brahmi has demonstrated promising efficacy in the management of various neuropharmacological conditions, including insomnia.

Table 1: Pharmacological activities and mechanisms of *Bacopa monnieri*.

S. No.	Pharmacological Activity	Mechanism of Action	Reference
1	Analgesic	Brahmi may also inhibit the nociceptive pathways (the pathways through which pain signals are transmitted), thereby reducing the sensation of pain. This effect is possibly mediated through the modulation of pain-related neurotransmitters and receptors. *Bacopa monnieri* is thought to promote the activity of the GABA system. It is an inhibitory neurotransmitter that helps to calm neuronal activity. Brahmi influences the echelons of various neurotransmitters in the brain. By modulating these neurotransmitters, Brahmi can enhance mood and reduce the perception of pain	[45-46].
2	Antidepressant	Important neurotransmitters that are involved in mood regulation are influenced by Brahmi. Enhancing mood and mitigating depressive symptoms, Brahmi increases the availability of these neurotransmitters. Cognitive impairment is often associated with depression. Brahmi's cognitive-enhancing properties can help improve memory, attention, and executive function, which can indirectly alleviate depressive symptoms.	[47-48]
3	Anticancer	To eradicate malignant cells without harming healthy cells, research indicates that Brahmi acts on specific pathways that cause cell death. Key players in apoptosis include caspases and other pro-apoptotic proteins. Brahmi has been found to inhibit angiogenesis, thereby starving tumors of the necessary nutrients and oxygen needed for their growth. Oxidative stress can damage DNA and other cellular components, leading to mutations and cancer development. By reducing oxidative stress, Brahmi can help protect cells from becoming cancerous.	[49]
4	Antiepileptic	Brahmi has the potential to affect the activity of ion channels, including calcium and sodium channels, which are essential for the production and transmission of electrical signals in the brain. Modulation of these channels can help stabilize neuronal activity and prevent seizures. Strong antioxidant qualities found in brahmi shield neurons from oxidative stress, which can heighten seizure activity.	[47-48, 50]
5	Antidiabetic	It has been demonstrated that Brahmi lowers blood glucose levels via several methods. It might raise insulin sensitivity, boost the activity of enzymes involved in glucose metabolism, and increase the uptake of glucose by cells.	[51]
6	Anti-inflammatory	Brahmi inhibits the synthesis and release of cytokines. These cytokines are implicated in a number of inflammatory illnesses and are essential in the inflammatory response. One important modulator of inflammation, the NF-κB pathway, can be inhibited by brahmi. Numerous genes involved in the inflammatory response are regulated by NF-κB. Brahmi lessens the synthesis of pro-inflammatory cytokines and enzymes by blocking this route.	[52-53]
7	Antimicrobial	The rupture of fungal cell membranes and the suppression of fungal enzyme activity are the mechanisms by which brahmi exerts its antifungal effect.	[54-55]

6. *Bacopa monnieri's* Mode of Action in Neurodegenerative Diseases

6.1 Alzheimer's Disease

Bacopa monnieri (Brahmi) exerts neuroprotective effects in Alzheimer's disease (AD) primarily by inhibiting acetylcholinesterase (AChE), the enzyme responsible for breaking down acetylcholine (ACh). By preventing ACh degradation, Brahmi helps maintain higher acetylcholine levels, which are critical for memory and cognitive function, particularly in AD, where cholinergic neurotransmission is compromised [56]. The potent antioxidant activity of Brahmi scavenges free radicals and mitigates oxidative stress, a major contributor to neuronal damage and cognitive decline in AD [57]. Additionally, Brahmi may reduce the formation and accumulation of amyloid-β plaques, which are neurotoxic and accelerate disease progression [58]. Brahmi also modulates the levels and activity of various neurotransmitters, improving mood, reducing anxiety, and enhancing overall cognitive function [59]. Neuroprotection is further supported by promoting neuronal repair, regeneration, and synaptic plasticity, essential for learning, memory, and cognitive function [60]. Some studies also suggest that Brahmi can chelate metal ions, such as iron and copper, which contribute to oxidative stress and amyloid plaque formation, thereby reducing their neurotoxic effects [61].

6.2 Parkinson's Disease

Parkinson's disease (PD) is characterized by dopaminergic neuronal loss and accumulation of α-synuclein, resulting in motor deficits and cognitive impairment. In animal models induced by rotenone, *Bacopa monnieri* demonstrated superior neuroprotective effects compared to levodopa [23, 62]. The neuroprotective mechanisms include modulation of catecholamine levels, which are crucial for normal brain function [63]. Its antioxidant and anti-inflammatory properties reduce reactive oxygen species (ROS), α-synuclein, and pro-inflammatory cytokines, mitigating the oxidative stress and neuroinflammation underlying PD. *Bacopa monnieri* also enhances endogenous antioxidant enzyme levels, such as glutathione peroxidase (GPx), catalase (Cat), and superoxide dismutase (SOD), thereby preventing oxidative damage and lipid peroxidation induced by neurotoxins like MPTP [50, 64]. These collective effects suggest that Brahmi may be more effective than conventional therapies like levodopa in reducing both the pathological features and symptoms of PD.

6.3 Epilepsy

Epilepsy, a chronic central nervous system disorder, is associated with deficits in cognition, learning, and memory. It involves abnormalities in GABAergic, glutamatergic, and cholinergic neuronal regulation [28]. Studies investigating the effects of *Bacopa monnieri* extract in pilocarpine-induced epilepsy models demonstrated significant downregulation of the NMDA receptor subunit NR1 in the hippocampus, restoring glutamate receptor regulation and potentially preventing excitotoxicity [52, 65-66]. Moreover, *Bacopa monnieri* modulates serum levels of T3, insulin, malate dehydrogenase, and acetylcholinesterase, which are linked to reduced peripheral nervous system dysfunction and seizure prevention [67]. These findings support the therapeutic potential of Brahmi and its constituents, particularly bacoside A, in epilepsy through mechanisms involving enhanced peripheral nervous system function, neuroprotection, and neurotransmitter regulation.

7. Clinical Evidence

Several clinical studies have evaluated the effects of *Bacopa monnieri* and its bioactive constituents on human cognitive health, memory, and neuropharmacological conditions, as summarized in **Table 2**. These studies provide evidence for its safety, efficacy, and potential as a complementary therapeutic agent in managing neurodegenerative and cognitive disorders.

Table 2: Clinical studies of Brahmi.

Participant Study	Study Design	Intervention	Clinical Outcomes	Ref.
Thirty-five healthy participants (75 percent female), 60–78 years old.	Open-label research, USA.	Participants get 320 mg of BM (CDRI 08) twice a day for three months.	After three months, there were no significant changes in the GDS or MoCA; a considerable improvement was seen in the delayed recall subscale. Significant	[68]

			decreases in NF-κB phosphorylation and increases in CREB phosphorylation were observed.	
Twenty Parkinson's disease volunteers (seven women and thirteen males, ages 69 to 85).	Primary, parallel, DB, controlled, interventional clinical trial. Brazil	For 90 days, patients were given either a placebo or BM (225 or 450 mg/day). Before, throughout, and ninety days following therapy, movement was assessed using the PDQL questionnaire.	The emotional function demonstrated time-dependent improvements, as shown by the delta percent (Δ%).	[69]
112 boys between the ages of 6 and 14 who were hyperactive and inattentive in comparison to a placebo, with ninety-three databases available for analysis.	This 14-week study was a DB, randomised, placebo-controlled experiment with an additional placebo run-in and run-out phase. Australia	Exceeding 14 weeks, patients were given 160 mg of BM (CDRI 08®) or a placebo (body weight 20–35 kg) or 320 mg/day (body weight exceeding 35 kg) of BM.	There were no discernible behavioural differences between the groups. Children who received BM made fewer mistakes and had faster reaction times. Interpersonal issues, executive functioning, cognitive flexibility, and sleep patterns all showed notable improvements.	[70]
48 Thai-ethnic patients, aged 55-80 years, who are in good health, can read and write in Thai.	Randomised, DB, PC investigation. Thailand.	During the placebo run-in period, each subject got one bottle of placebo every two weeks. Next, participants were split into two groups: one for treatment (1 bottle of BM essence daily for 12 weeks) and one for placebo.	Memory speed was enhanced by BM essence (assessed by computerised tests).	[71]
After four weeks of citalopram treatment, 42 patients (64% women; >47 years old) with serious depression (DSM 5.0) and a considerable degree of anhedonia (SHAPS score ≥ 3) did not show a meaningful response.	Randomized study Italy	For four weeks, patients were given either citalopram or citalopram combined with BM (300 mg).	Notable improvements were found on the Hamilton depression rating scale, SHAPS, and Strengths and Challenges Questionnaire.	[72]
48 AD patients, along with their excellence of life and cognitive results.	Phase 2b Randomised DB, Parallel Study, India.	For a year, patients were given either 300 mg of BM or 10 mg of donepezil.	There was no discernible variation in the memory scale's rate of change at three, six, and nine months. There was a notable variation in the PGIMS score change between BM and donepezil in the most recent follow-up.	[73]

80 healthy participants, 25 men and 55 women, aged 60 or over.	DB, parallel arm, controlled, and random study design. Italy.	Following a one-week baseline, participants were given either a daily placebo or a combination of antioxidants for eight weeks.	The mix can be consumed to prevent the cognitive discount associated with brain ageing, as seen by improvements in TMT scores and verbal fluency test evaluations of letter fluency.	[74]
Twelve patients with dementia older than eighteen.	Pilot study.	Every grade of dementia participants received 250 mg of BM every three months.	Every patient responded well; GDS was 4.42 before BM dealing and 1.92 following three months.	[75]
Thirty senior participants, ages 61–69, self-perceived cognitive deterioration, and basal MMSE scores 20–27.	DB, a crossover experiment comparing a placebo Outpatient clinical practice setting. Italy	The participants were given a combination of nutraceuticals.	In both groups, there was a notable upgrade in the SRDS scores. The active therapy arm saw a considerable improvement in the MMSE and PSQ Index.	[76]
Sixty 19–22-year-old healthy medical Students.	A similar, non-crossover, randomised PC experiment was carried out outside. India	The subjects were given 150 mg of BM (Bacognize®) or a placebo twice a day for six weeks.	After BM was administered, there was a significant improvement in cognitive performance as well as a notable increase in serum calcium.	[77]
109 individuals in good health and 123 SDAT sufferers older than 60109 individuals in good health and 123 SDAT sufferers older than 60.	DB, PC, randomised clinical trial with active control. India	Four groups of participants: In Group C, patients were treated with donepezil; Group D patients were treated with the test formulation; Group A consisted of strong aging subjects who received a placebo; Group B consisted of strong aging subjects who received the test formulation.	When BM was administered to healthy aged adults, as opposed to a placebo, and to SDAT patients, as opposed to donepezil, the results showed a significant increase in cognitive abilities as well as a reduction in neurodegenerative syndromes.	[78]
An open-label clinical trial was carried out in India.	31 children, 3 women and 28 men, aged 6 to 12, had an age of onset of ADHD before 7 years.	For six months, patients were given 225 mg of BM extract (BacoMind ®) daily.	ADHD symptoms were significantly reduced in scores, except for social problems.	[79]
Australia-based double-blind crossover trial with placebo control.	17 well patients, ages 18 to 44, 13 women and 4 males17 well patients, ages 18	After taking a placebo, patients were given 320 mg of BM and 640 mg of BM one hour and two hours later.	There were improvements in mood and cognition, and cortisol levels dropped.	[80]

	to 44, 13 women and 4 males.			
Australia; DB, PC.	Twenty women and four men, ages 18 to 56, who were in good health, finished a battery of mentally taxing tasks.	In a cross-over design, patients were given either 320 mg or 640 mg of BME (KeenMind®-CDRI 08).	There was no effect on CV activity or task-induced feelings of fatigue and tension.	[81]
DB, randomised, PC setup, Thailand.	60 healthy adults, 23 males and 37 women, with a mean age of 62.62 years.	Individuals were given either BM (300 and 600 mg) or a placebo once every 12 weeks.	Working memory as well as improvements in attention, working memory, and cognitive processing.	[82]
DB, PC, randomised experiment, Australia.	98 fit participants, 46 men and 52 women, ages 55 to 58, were recruited.	For 12 weeks, patients were given either 300 mg/day of BM (BacoMin) or a similar placebo.	Both the acquisition and retention of memories improved.	[83]
DB, PC, randomised study, Australia (with a 6-week placebo run-in)	54 patients, 65 years of age or older, without clinical indications of dementia (30% men and 60% women).	For 12 weeks, patients were given 300 mg/day of BM extract or a comparable placebo.	The AVLT delayed word recall memory scores improved. Heart rate, anxiety, and depression all dropped.	[18]
Children in the CRMR, Mumbai, India, who require individualised educational help for 10.5 years.	The trial was carried out under strict observation as an outpatient surgery in a hospital.	225 milligrams of bacopa extract every day for 16 weeks.	Working memory and Short-term verbal memory, logical memory, personal life memory, and both visual and auditory memory showed notable improvement ($P \leqslant .05$).	[84]
65 people, aged 50 to 75, who have had a memory impairment for at least a year based on their report (MMSE > 24). At 0, 12 and 24 weeks, neuropsychological evaluations were conducted. Results were examined in terms of attention, memory, and information processing speed.	DB, randomised, PC study. India.	In a 24-week research, patients were given BacoMind® 450 mg/day for 12 weeks or a placebo, plus a further 12 weeks of placebo.	On the verbal memory and focus tests that were looked at, BM enhanced performance.	[85]

Randomised, DB, PC experiment. Australia.	107 well-aged persons, ages 18 to 60, of whom 62 finished the study (21 men and 41 women).	The subjects were given either a placebo or BM. Assessments of neuropsychology were carried out at 0 and 90 days.	BM significantly increased performance on the "working memory"	[86]
Forty patients above the age of fifty-five reported experiencing memory loss during routine tasks (WMS: logical subset score < 6).	Randomised, DB, PC, trial conducted in India	For 12 weeks, patients received either 125 mg of BM extract or a placebo twice a day; the placebo was administered to both groups for an extra 4 weeks.	Reasonable memory, mental control, and paired related learning all significantly improved with BM.	[87]
76 fit adults, ages 40–65, 48 women and 28 males.	Australia: DB, randomised, PC trial	Three months' supply of capsules (450 mg for individuals over 90 kg and 300 mg for those under 90 kg) is administered during the first session. Second session: Patients were told not to take the capsules three months later. The trial session finished, and the third one started six weeks later.	The retention of new information was significantly impacted. Some factors remained unchanged.	[88]
46 fit volunteers, 35 women and 11 men, ages 18 to 60.	DB, independent-group, PC study. Australia	Participants received either a placebo or a combination of bacosides A and B, or both, for 12 weeks.	The rate of learning, the pace at which visual information is processed, and memory consolidation all showed notable improvements.	[46]
Healthy Children from Rural India, Ages 6-8.	An independent group study that was DB, PC, and randomized.	Bacopa syrup, one teaspoonful 3 times a day for 3 months.	Enhanced perceptual organisation and reasoning ability, enhanced perceptual representations of patterns, enhanced exploration drive (as assessed by maze learning), and improved reaction time	[89]

8. Conclusion

The potential therapeutic effects of *Bacopa monnieri* (Brahmi) in the management of neurodegenerative disorders have garnered considerable attention. Numerous clinical trials have demonstrated that Brahmi can enhance cognitive functions, including memory, attention, and learning. These effects are particularly relevant in conditions such as Alzheimer's disease, where cognitive decline is a hallmark symptom. Brahmi has been shown to modulate key neurotransmitters, including acetylcholine, dopamine, and serotonin, which may contribute to improved neuronal communication and overall brain health. Its neuroprotective mechanisms are multifaceted, involving the upregulation of neurotrophic factors, inhibition of amyloid-β aggregation, and enhancement of synaptic plasticity. Consequently, *Bacopa monnieri* represents a promising complementary

therapeutic strategy that could be integrated into more comprehensive approaches for the treatment of complex neurodegenerative disorders.

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How to cite this article: Jyoti, Mukhija M, Kamboj S, Sharma D. Brahmi and its Extracts in the Administration of Neurodegenerative Syndromes. Pharm Res Bull. 2026;5(1):109-123.