

# Ashwagandha (*Withania somnifera*): Herbal Remedies in Neurodegenerative Disorders

Dinesh Kumar<sup>1</sup>, Prteek Kaur<sup>1</sup>, Neelam Sharma<sup>1\*</sup>, Punam Gaba<sup>1</sup>, Rahul Kumar Sharma<sup>1</sup>, Shailesh Sharma<sup>1</sup>.

<sup>1</sup> Amar Shaheed Baba Ajit Singh Jujhar Singh Memorial College of Pharmacy, Bela, Ropar-140111, Punjab, India.

\* Correspondence: pharmneelam@gmail.com

Received: 18 October 2025; Accepted: 07 January 2026; Published: 19 January 2026

**Abstract:** Ashwagandha (*Withania somnifera*) is a perennial medicinal shrub, renowned for its therapeutic properties, particularly those of its roots. The name “Ashwagandha” is derived from the Vedic Sanskrit words “ashwa,” meaning “horse,” and “gandha,” meaning “scent,” reflecting its traditional association with vitality and strength. Also referred to as Indian ginseng, Ashwagandha thrives in arid regions across the Mediterranean belt, India, Sri Lanka, Africa, and Pakistan. Ashwagandha is distinguished by its neuroprotective potential, with growing evidence suggesting its efficacy against neurodegenerative disorders such as Alzheimer’s and Parkinson’s disease. These neuroprotective effects are primarily attributed to its active constituents, particularly the withanolides, which exhibit potent antioxidant activity by inhibiting the formation of harmful free radicals. By reducing oxidative stress, a key driver of neurodegeneration, Ashwagandha helps preserve neuronal integrity. In addition, Ashwagandha exerts anti-inflammatory effects by modulating pro-inflammatory pathways, including the Nuclear Factor-kappa B (NF-κB) signaling pathway, which is implicated in the progression of neurodegenerative diseases. It also promotes neurogenesis and enhances synaptic plasticity by upregulating brain-derived neurotrophic factor (BDNF) and other neurodevelopmental factors critical for neuronal survival and function. Experimental studies have demonstrated that Ashwagandha alleviates neuronal damage, mitigates oxidative stress, supports mitochondrial function, and improves cognitive performance. Its adaptogenic properties further stabilize the hypothalamic-pituitary-adrenal (HPA) axis, reducing chronic stress, which is detrimental to neuronal health. Collectively, these synergistic effects position Ashwagandha as a promising natural therapeutic agent for enhancing cognitive function, protecting neurons, and potentially slowing the progression of neurodegenerative disorders.

**Keywords:** Ayurvedic medicine, Phytochemicals, Anti-inflammatory, Neuroprotective, Adaptogenic, Antioxidant effects, *Withania somnifera*.

## 1. Introduction

Ashwagandha, commonly known as Indian winter cherry and also referred to as Indian ginseng, is scientifically identified as *Withania somnifera*. The primary medicinal part of the plant is its root. The term “Ashwagandha” originates from Sanskrit, derived from the words “ashwa,” meaning horse, which symbolizes the strength imparted by the root, and “gandha,” referring to the distinctive odor of the root [1]. The genus *Withania* comprises 26 species, which are naturally distributed across South Asia and the Eastern Mediterranean region [2]. Until recently, only two species—*Withania somnifera* and *Withania coagulans*—were considered native to India. However, a third species, *Withania ashwagandha*, has now been identified in Indian germplasm [3].

**Common name:** Indian Ginseng, Winter Cherry, Asgandh, Nagouri or Punir

**Scientific name:** *Withania somnifera*

**Family:** Solanaceae

**Origin:** India

**Chromosome Number:**  $2n=48$

**Productive part:** Roots, Leaves and Seeds

**Geographical Source:** *Withania somnifera*, commonly known as Indian ginseng, Suranjan, winter cherry, or Ashwagandha, is a hardy plant prized for its extensive therapeutic applications and pharmaceutical benefits. Adapted to arid environments, it grows abundantly across the Mediterranean belt, Africa, India, Sri Lanka, and Pakistan [4].

### 1.1 Botanical Description

*Withania somnifera* is a robust, perennial shrub exhibiting high drought tolerance. It typically grows to a height of approximately 2 meters with a spread of about 1 meter. The plant is characterized by short hairs, giving it a silvery-grey appearance. Leaves are elliptical, green on the upper surface, and densely hairy on the underside. The plant produces small, green, bell-shaped flowers, usually 1–7 per leaf node, each with a calyx approximately 5 mm long, expanding up to 20 mm in fruit. The corolla is narrow and yellowish-green, bearing prominent yellow-orange stamens. Fruits are smooth, round berries enclosed by a papery calyx, measuring 5–8 mm in diameter, initially green and turning red upon ripening. The roots are fleshy and robust, emitting a strong odor and possessing a bitter taste, with secondary branches resembling fibers [5].

### 1.2 Historical and Traditional Use

Ayurveda, the ancient Indian system of medicine, originated around 6000 BC [6]. For centuries, Ashwagandha has been recognized as a potent Rasayana, standing out among Ayurvedic rejuvenating herbs. Classified as a "Sattvic Kapha Rasayana," the root is highly valued for its restorative, libido-enhancing, sedative, diuretic, deworming, astringent, thermogenic, and invigorating properties. The name Ashwagandha, meaning "scent of a horse," reflects its distinctive aroma and the traditional belief that it imparts the strength of a horse.

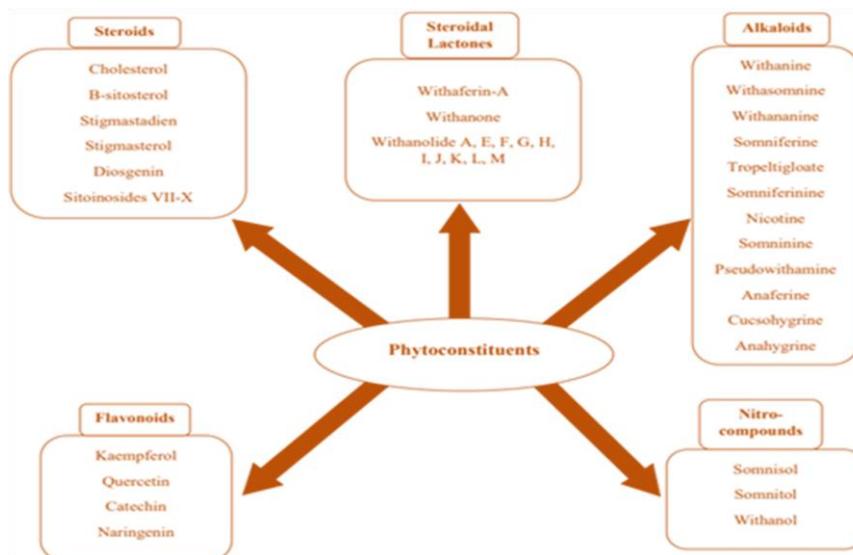
Traditionally, Ashwagandha has been employed to treat conditions such as child malnutrition (particularly when administered with milk), age-related weakness, rheumatism, disorders associated with vata imbalance, leucoderma, constipation, insomnia, nervous breakdowns, and goiter [7]. A root decoction with water is used to alleviate joint inflammation and treat sores, boils, and swellings [8-9]. Combined with other remedies, it is also recommended for snake bites and scorpion stings, as well as for pimples, leucorrhea, flatulent colic, worms, boils, and piles [10]. Among various Ashwagandha types, Nagori Ashwagandha is often considered the most efficacious, with freshly ground root powder yielding the greatest therapeutic benefits [11].

The foliage, which is bitter in taste, is used to treat fever and painful inflammations. Flowers possess tissue-firming, detoxifying, diuretic, and libido-enhancing properties. Seeds are employed as anthelmintics and, when combined with tissue-constricting compounds and rock salt, can remove white patches from the cornea. Ashwagandha Rishta, a preparation derived from the plant, is used to manage hysteria, anxiety, memory loss, fainting spells, and other conditions, and is also regarded as a tonic for enhancing sperm production [7].

## 2. Phytochemical Properties

The identification and characterization of secondary metabolites are critical for developing novel therapeutic agents [12]. Various phytochemicals have been isolated from *Withania somnifera* using advanced analytical techniques such as column chromatography, LC-MS, X-ray diffraction, NMR, and GC-MS [5, 13-14]. Phytochemical investigations have revealed a diverse array of bioactive compounds in different parts of the plant. Key constituents include saponins, alkaloids, flavonoids, steroidal lactones, tropanol, starch, tannins, phenolic compounds, somniferinine, scopoletin, carbohydrates,  $\beta$ -sitosterol, 6,7 $\beta$ -epoxywithanone, cysteine, 14- $\alpha$ -hydroxywithanone, anaferine, withanolides, sitoindosides, anahygrine, cuscohygrine, pseudotropine, withanine, withananine, somniferiene, and chlorogenic acid [4, 15]. Alcoholic tinctures of the leaves and roots have yielded additional organic compounds, including somnitol, somnisol, withaniol, withanic acid, and phytosterols, along with alkaloids such as withananine, somniferinine, withanine, somniferine, pseudowithamine, and withasomnine (**Figure 1**). These bioactive metabolites are largely responsible for the plant's therapeutic and pharmacological effects. The first withanolide isolated from *Withania somnifera* was Withaferin-A [16]. Subsequent research has identified several other withanolides, including Withanolide-A, Withanolide-E, and Withanone, among others [17]. Methanolic leaf extracts have revealed the presence of visamine, tisopelletierine, cuscohygrine, 3 $\alpha$ -tigloyloxytropine, hentriacontane, and various other compounds [18]. Phytochemical analyses further indicate the presence of reducing sugars, iron, dulcitol, starch, and

essential amino acids such as cysteine, tryptophan, and glutamic acid. Additionally, sterols including cholesterol, diosgenin, stigmastadiene, and sitoindosides VII–X have been identified [19]. Seven new withanoside glycosides (withanosides I–VII) were also isolated, alongside four known compounds — coagulin Q, 5 $\alpha$ ,20 $\alpha$ F (R)-dihydroxy-6 $\alpha$ ,7 $\alpha$ -epoxy-1-oxowitha-2,24-dienolide, physagulin D, and Withaferin-A.



**Figure 1:** *Withania somnifera* contains a variety of chemical substances.

### 3. Pharmacological Properties

Centuries of Ayurvedic knowledge, complemented by modern research, have demonstrated that *Withania somnifera* exhibits a wide range of pharmacological activities, including adaptogenic, antimicrobial, abortifacient, libido-enhancing, astringent, anti-inflammatory, diuretic, sedative, and invigorating effects. It also exerts potent antioxidant activity [20-21], enhances immune function by stimulating lymphocytes and phagocytes [22-23], and helps mitigate stress, promoting overall well-being.

#### 3.1 Anti-Inflammatory Activity

Withaferin-A, a major active steroid in Ashwagandha, exhibits strong anti-inflammatory and anti-arthritic properties. Studies indicate that it is comparable in efficacy to hydrocortisone sodium succinate at equivalent doses [24]. Remarkably, Withaferin-A alleviated arthritic symptoms in animal models without adverse effects, including weight loss observed with hydrocortisone. In adjuvant-induced arthritis in rats, doses of 12–25 mg/kg intraperitoneally significantly reduced swelling, with effects persisting for at least four hours [25]. *Withania somnifera* has also demonstrated anti-inflammatory activity in models of cotton pellet granuloma, carrageenan-induced inflammation, and formaldehyde-induced arthritis, by modulating acute-phase responses and reducing serum  $\beta$ -1 globulin levels [26].

#### 3.2 Antibiotic Activity

Both roots and leaves of Ashwagandha possess antimicrobial properties. Withaferin-A (10  $\mu$ g/mL) inhibited the growth of Gram-positive bacteria, acid-fast bacilli, and pathogenic fungi. It was effective against *Micrococcus pyogenes* var. aureus and partially inhibited *Bacillus subtilis* glucose-6-phosphate dehydrogenase activity. Withaferin-A also displayed antiviral activity against Ranikhet virus, *Entamoeba histolytica*, and Vaccinia virus, and protected against systemic *Aspergillus* infection by enhancing macrophage phagocytic activity in mice. Its antibiotic effects are attributed to the unsaturated lactone ring structure, demonstrating superior efficacy in experimental abscesses in rabbits compared to penicillin [27].

#### 3.3 Anti-aging Activity

In a double-blind, placebo-controlled study involving 101 men aged 50–59, daily administration of 3 g of Ashwagandha for one year resulted in improvements in hemoglobin levels, hair pigmentation, red blood cell

count, and standing height. Blood cholesterol levels decreased, calcium levels in fingernails remained stable, and 70% of participants reported enhanced sexual function [28].

### 3.4 Anticonvulsant Activity

Ashwagandha root extract reduced spasms and twitching in 70% and 10% of treated subjects, respectively, at 100 mg/kg. It also decreased the severity of PTZ-induced seizures, as evidenced by EEG changes [29], and reduced motor seizure severity induced via electrical stimulation in the basolateral amygdala. The anticonvulsant effects are suggested to be mediated through GABAergic pathways [30].

### 3.5 Immunomodulatory Activity

Ashwagandha significantly modulated immune function in animal models. In mice treated with immunosuppressive drugs (cyclophosphamide, azathioprine, and prednisolone), Ashwagandha prevented bone marrow suppression and enhanced hemoglobin, RBC, platelet counts, and body mass [31]. It also countered reductions in WBC counts due to cyclophosphamide and gamma radiation and increased  $\beta$ -esterase-positive bone marrow cells. Withanolide-E and Withaferin-A selectively inhibited human B and T cells, as well as murine thymocytes, demonstrating targeted immunomodulation [32].

### 3.6 Anti-hyperglycaemic Effect

In combination formulations such as Transina, Ashwagandha reduced streptozotocin (STZ)-induced hyperglycemia in rats, likely by scavenging free radicals in pancreatic islets. STZ typically induces hyperglycemia via reduced superoxide dismutase (SOD) activity in  $\beta$ -cells, leading to oxidative damage.

### 3.7 Other Therapeutic Benefits

Ashwagandha has shown therapeutic potential in osteoarthritis, inflammation, stroke, and tardive dyskinesia. It exhibits antimicrobial activity, including antifungal effects and moderate antibacterial activity against *Staphylococcus aureus* and *Pseudomonas aeruginosa* [33].

## 4. Clinical Evidence for Neuroprotective Effect

Multiple studies have established that *Withania somnifera* exhibits significant neuroprotective effects [34-38]. Both leaf extracts and the bioactive compound withanone have been shown to protect neurons and glial cells from scopolamine-induced damage. Specifically, *W. somnifera* markedly attenuated the adverse effects of scopolamine on key neuronal markers such as GAP-43, MAP-2, NF-H, PSD-95, and the glial marker GFAP. It also reduced scopolamine-induced DNA damage and oxidative stress [39]. Moreover, components of *W. somnifera* alleviated lead-induced cytotoxicity in neuroglial cells by modulating heat shock proteins (HSP70), GFAP expression, mortalin, and neural cell adhesion molecule (NCAM) [40]. Glycowithanolides exhibited potent antioxidant activity in the cerebral cortex and striatum of rat brains, enhancing catalase (CAT), superoxide dismutase (SOD), and glutathione peroxidase (GPx) activity in a dose-dependent manner [41]. Additionally, *W. somnifera* extracts protected streptozotocin-treated mice from oxidative damage by reducing oxidative stress [42] and significantly decreased neuronal degeneration in the CA2 and CA3 hippocampal regions in immobilization stress-induced rats [43].

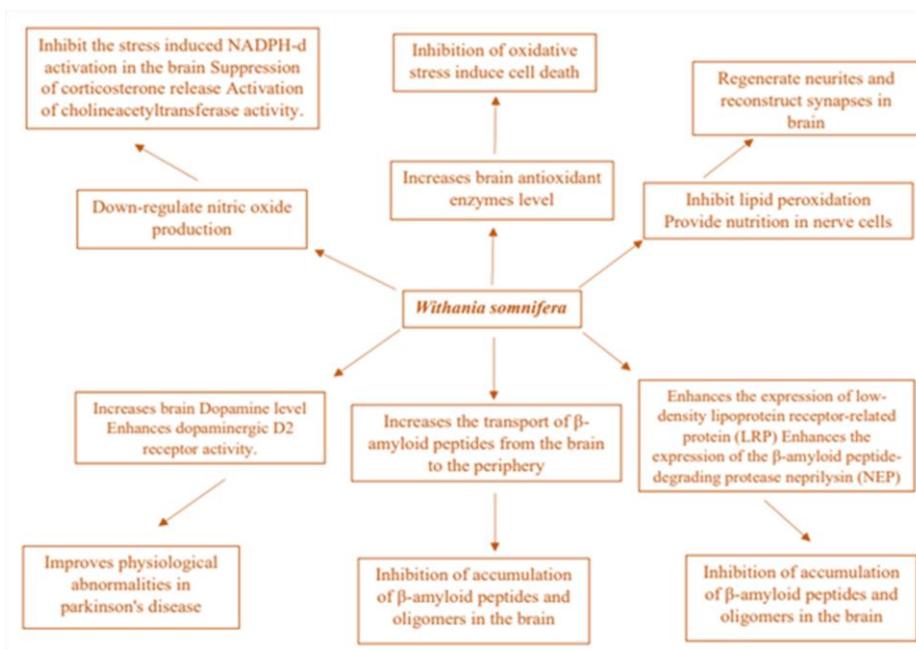
Root-derived compounds of *W. somnifera* have been shown to stimulate neurite outgrowth in human neuroblastoma cell cultures, with Withanolide-A promoting axonal growth, while Withanolides-IV and VI primarily enhanced dendritic growth. Withanoside-IV facilitated both dendrite and axon regeneration and repaired synapses in rat cortical neurons impaired by amyloid-beta ( $A\beta$ ). Furthermore, leaf extracts protected retinoic acid-differentiated C6 and IMR-32 cells from glutamate-induced excitotoxicity, alleviating stress responses by increasing HSP70 levels and restoring neuronal plasticity, as indicated by neural cell adhesion molecules and their polysialylated forms [44]. *W. somnifera* also mitigates excitotoxic damage induced by kainic acid through the reduction of oxidative stress. In models of middle cerebral artery occlusion (MCAO), it reduced oxidative stress, lesion volume, and improved functional outcomes [45]. In mouse MCAO models, it decreased infarct size by modulating heme oxygenase-1 and the pro-apoptotic protein PARP-1 via the AIF pathway, while also reducing Semaphorin-3A expression to facilitate recovery [46-47]. Withanone and Withanolide-A were shown to upregulate anti-apoptotic proteins and downregulate pro-apoptotic proteins.

Withanolide-A conferred neuroprotection in ischemia-reperfusion injury by decreasing infarct size, preventing blood-brain barrier breakdown, reducing brain edema, and modulating neurotransmitter release [48]. Withaferin-A attenuated neuroinflammation and misfolded SOD1 in amyotrophic lateral sclerosis (ALS), slowing disease progression and prolonging lifespan [49].

In addition to neuroprotection, *W. somnifera* exhibits anxiolytic and antidepressant effects comparable to Lorazepam and Imipramine [43]. Glycowithanolides demonstrate anxiolytic activity similar to conventional antidepressants [50]. Polyherbal formulations such as Perment® have also shown anxiolytic and antidepressant effects mediated via serotonergic and adrenergic pathways [51].

## 5. Mechanism of Action in Neurodegenerative Disorders

Ashwagandha, a revered herb in Ayurvedic medicine, exerts neuroprotective effects through multiple mechanisms targeting the underlying causes of neurodegeneration. Its primary action involves modulation of the hypothalamic-pituitary-adrenal (HPA) axis, reducing chronic stress that is deleterious to neurons. Withanolides, the principal bioactive compounds, possess potent antioxidant properties, which counteract oxidative stress by scavenging harmful free radicals and enhancing endogenous antioxidant enzymes, including superoxide dismutase and catalase. *Withania somnifera* also promotes neuronal survival, synaptic plasticity, and repair of damaged neurons, contributing to its therapeutic potential in neurodegenerative disorders (Figure 2).



**Figure 2:** Neuroprotective mechanisms of *Withania somnifera*.

Ashwagandha supports cognitive function by promoting neurogenesis and enhancing synaptic plasticity, partly through increasing levels of brain-derived neurotrophic factor (BDNF) and other growth factors critical for neuronal survival. Its anti-inflammatory effects are significant, suppressing pro-inflammatory pathways, including NF- $\kappa$ B (Nuclear Factor-kappa B), which are implicated in the pathogenesis of neurodegenerative diseases. Through a combination of antioxidant, anti-inflammatory, neurogenic, and stress-reducing mechanisms, Ashwagandha protects neurons, enhances cognitive performance, and may slow the progression of disorders such as Parkinson's and Alzheimer's disease [52-53].

## 6. Applications of Ashwagandha

Ashwagandha, commonly referred to as Indian ginseng, is a perennial shrub with a wide spectrum of potential medicinal applications. The major therapeutic uses are outlined below:

### 6.1 Neuroprotective and Neurodegenerative Effects

#### 6.1.1 Use in Alzheimer's Disease

Alzheimer's disease is characterized by the accumulation of  $\beta$ -amyloid proteins in the brain, which, in fibrillar form, are neurotoxic. These proteins induce oxidative stress, inhibit glucose transport, and ultimately lead to neuronal death. Ashwagandha has been shown to suppress  $\beta$ -amyloid-induced cytotoxicity and improve neurocognitive outcomes. Specifically, withaferin A, a bioactive compound from Ashwagandha, can inhibit  $\beta$ -amyloid aggregation and prevent  $\tau$  protein accumulation, positioning it as a promising therapeutic candidate for Alzheimer's disease [54].

### 6.1.2 Use in Parkinson's Disease

Parkinson's disease involves the degeneration of dopaminergic neurons in the nigrostriatal pathway, leading to reduced inhibitory dopamine signaling and increased excitatory input from neurotransmitters such as acetylcholine and glutamate. In rat models of 6-hydroxydopamine-induced Parkinson's disease, Ashwagandha treatment significantly decreased lipid peroxidation, increased glutathione levels, and enhanced the activities of antioxidant enzymes, including superoxide dismutase, glutathione peroxidase, glutathione S-transferase, catalase, and glutathione reductase. Additionally, it increased catecholamine levels, dopamine D2 receptor binding, and tyrosine hydroxylase expression, highlighting its potential as a neuroprotective and dopaminergic-enhancing agent [55].

## 6.2 Anticancer Effects

Ashwagandha (*Withania somnifera*) is a rich source of bioactive compounds found in its roots, stems, and leaves, many of which exhibit significant anticancer properties. Key constituents, including withanolides and withaferin A, have been shown to inhibit the growth and proliferation of various cancer cell lines by inducing apoptosis, cell cycle arrest, and inhibition of angiogenesis. These compounds can act independently or synergistically with standard chemotherapeutic agents, enhancing their efficacy while potentially reducing side effects. Preclinical studies suggest that Ashwagandha-derived molecules modulate multiple signaling pathways involved in cancer progression, highlighting their promising role as complementary or adjuvant therapies in oncology [56].

## 6.3 Antidiabetic Activity

Withaferin A has been shown to regulate experimentally induced type 1 diabetes in rats via modulation of the Nrf2/NF $\kappa$ B signaling pathway, demonstrating therapeutic potential. Computational (in silico) studies further support Withaferin A as a candidate for diabetes therapy, though clinical trials in humans are required to confirm safety and efficacy [57].

## 6.4 Anti-Inflammatory Effects

Ashwagandha demonstrates potential in managing inflammatory diseases, including cardiovascular, pulmonary, autoimmune disorders, diabetes, cancers, and neurodegenerative conditions. Preclinical studies indicate that Ashwagandha modulates mitochondrial metabolism, apoptosis, and reduces inflammation by downregulating pro-inflammatory markers such as nitric oxide, IL-6, TNF- $\alpha$ , and reactive oxygen species. These findings suggest its promise as a natural therapeutic agent, pending validation in human clinical trials [58].

## 6.5 Treatment of Sleep Disorder

Insomnia, characterized by insufficient or disturbed sleep, affects daytime functioning. Clinical trials have shown that Ashwagandha root extract (300 mg twice daily for over 10 weeks) significantly improves sleep quality and reduces sleep onset latency. Studies in elderly participants (aged 65–80 years) further confirmed improvements in sleep quality, alertness, and overall well-being, with good tolerability and safety [59].

## 6.6 Other Applications of Ashwagandha

Ashwagandha is also recognized for its broad-spectrum health benefits. Emerging research suggests potential utility in COVID-19 management due to its immunomodulatory, anti-inflammatory, anti-stress, anti-diabetic, and antihypertensive properties. Compounds such as withanoside V and somniferin have shown inhibitory effects on the SARS-CoV-2 main protease, indicating potential antiviral applications [60].

## 7. Conclusion

Ashwagandha (*Withania somnifera*) has emerged as a potent natural neuroprotective agent owing to its diverse pharmacological properties. Its ability to counteract oxidative stress, reduce neuroinflammation, and enhance neurogenesis underscores its therapeutic potential in preserving cognitive function and mitigating neurodegenerative disorders such as Parkinson's and Alzheimer's disease. The bioactive compounds, particularly withanolides, mediate their antioxidant and anti-inflammatory activities, providing neuronal protection and maintaining overall brain health. Ashwagandha also exhibits adaptogenic properties, stabilizing the hypothalamic-pituitary-adrenal (HPA) axis and alleviating stress-induced neuronal impairment. Moreover, it enhances synaptic plasticity by increasing brain-derived neurotrophic factor (BDNF) levels, which is critical for neuronal survival and cognitive function. In addition, Ashwagandha supports mitochondrial integrity, ensuring efficient energy metabolism in neurons. By targeting multiple pathways implicated in neurodegeneration, Ashwagandha represents a promising complementary therapeutic option for neurodegenerative diseases. Although preclinical and clinical studies have shown encouraging results, further research is necessary to establish its long-term safety, efficacy, optimal dosing, and standardized preparations. Mechanistic studies will also be essential for fully elucidating its neuroprotective potential. Given its broad spectrum of benefits, Ashwagandha holds significant promise as a natural strategy to enhance cognition, improve mental resilience, and potentially slow the progression of neurodegenerative disorders.

## 8. References

1. Mikulska P, Malinowska M, Ignacyk M, Szustowski P, Nowak J, Pesta K, Szelağ M, Szklanny D, Judasz E, Kaczmarek G, Ejiohuo OP, Paczkowska-Walendowska M, Gościński A, Cielecka-Piontek J. Ashwagandha (*Withania somnifera*)-Current Research on the Health-Promoting Activities: A Narrative Review. *Pharmaceutics*. 2023;15(4):1057.
2. Khabiyi R, Choudhary GP, Jnanisha AC, Kumar A, Lal RK. An insight into the potential varieties of Ashwagandha (Indian ginseng) for better therapeutic efficacy. *Ecol Front*. 2024;44(3):444–450.
3. Matsuda H, Murakami T, Kishi A, Yoshikawa M. Structures of withanosides I, II, III, IV, V, VI, and VII, new withanolide glycosides, from the roots of Indian *Withania somnifera* DUNAL. and inhibitory activity for tachyphylaxis to clonidine in isolated guinea-pig ileum. *Bioorg Med Chem*. 2001;9(6):1499–1507.
4. Saleem S, Muhammad G, Hussain MA, Altaf M, Bukhari SNA. *Withania somnifera* L.: Insights into the phytochemical profile, therapeutic potential, clinical trials, and future prospective. *Iran J Basic Med Sci*. 2020;23(12):1501–1526.
5. Paul S, Chakraborty S, Anand U, Dey S, Nandy S, Ghorai M, Saha SC, Patil MT, Kandimalla R, Proćków J, Dey A. *Withania somnifera* (L.) Dunal (Ashwagandha): A comprehensive review on ethnopharmacology, pharmacotherapeutics, biomedicinal and toxicological aspects. *Biomed Pharmacother*. 2021;143:112175.
6. Wiciński M, Fajkiel-Madajczyk A, Sławatycki J, Szambelan M, Szyperski P, Wojciechowski P, Wójcicki J, Gawryjolek M. Ashwagandha (*Withania somnifera*) and Its Effects on Well-Being—A Review. *Nutrients*. 2025; 17(13):2143.
7. Sharma P. Ashwagandha, Dravyaguna Vijnana. Varanasi (India): Chaukhambha Vishwabharti; 1999. p. 763–765.
8. Bhandari C. Ashwagandha (*Withania somnifera*). Varanasi (India): CS Series of Varanasi Vidyavilas Press; 1970. Vol. 1. p. 96–97.
9. Kritikar K, Basu B. Indian medicinal plants. Allahabad (India): Lalit Mohan Basu; 1935. p. 1774–1776.
10. Misra B. Ashwagandha—Bhavaprakash Nighantu (Indian Materia Medica). Varanasi (India): Chaukhambha Bharti Academy; 2004. p. 393–394.
11. Singh N, Bhalla M, de Jager P, Gilca M. An overview on ashwagandha: a Rasayana (rejuvenator) of Ayurveda. *Afr J Tradit Complement Altern Med*. 2011;8(5 Suppl):208-13.
12. Nabi M, Tabassum N, Ganai BA. *Skimmia anquetilia* N.P. Taylor and Airy Shaw (Rutaceae): A Critical Appraisal of its Ethnobotanical and Pharmacological Activities. *Front Plant Sci*. 2022;13:930687.
13. Tong X, Zhang H, Timmermann BN. Chlorinated Withanolides from *Withania somnifera*. *Phytochem Lett*. 2011;4(4):411–414.
14. Tewari D, Chander V, Dhyani A, Sahu S, Gupta P, Patni P, Kalick LS, Bishayee A. *Withania somnifera* (L.) Dunal: Phytochemistry, structure-activity relationship, and anticancer potential. *Phytomedicine*. 2022;98:153949.
15. Power FB, Salway AH. The constituents of *Withania somnifera*. *J Chem Soc Trans*. 1911;99:490–507.

16. Lavie D, Glotter E, Shvo Y. Constituents of *Withania somnifera* Dun. Part IV. The structure of withaferin A. *J Chem Soc.* 1965;7517–7531.
17. Ali A, Maher S, Khan SA, Chaudhary MI, Musharraf SG. Sensitive quantification of six steroidal lactones in *Withania coagulans* extract by UHPLC electrospray tandem mass spectrometry. *Steroids.* 2015;104:176–181.
18. Afewerky HK, Ayodeji AE, Tihamiyu BB, Orege JI, Okeke ES, Oyejobi AO, Bate PNN, Adeyemi SB. Critical review of the *Withania somnifera* (L.) Dunal: ethnobotany, pharmacological efficacy, and commercialization significance in Africa. *Bull Natl Res Cent.* 2021;45(1):176.
19. Bharti VK, Malik JK, Gupta RC. Ashwagandha: multiple health benefits. In: Gupta RC, editor. *Nutraceuticals.* Elsevier; 2016. p. 717–733.
20. Abou-Douh AM. New withanolides and other constituents from the fruit of *Withania somnifera*. *Arch Pharm (Weinheim).* 2002;335(6):267–276
21. Panda S, Kar A. Evidence for free radical scavenging activity of ashwagandha root powder in mice. *Indian J Physiol Pharmacol.* 1997;41(4):424–426.
22. Wagner H, Nörr H, Winterhoff H. Plant adaptogens. *Phytomedicine.* 1994;1(1):63–76.
23. Singh B, Saxena AK, Chandan BK, Gupta DK, Bhutani KK, Anand KK. Adaptogenic activity of a novel, withanolide-free aqueous fraction from the roots of *Withania somnifera* Dun. *Phytother Res.* 2001;15(4):311–318.
24. Khare C. *Indian medicinal plants: an illustrated dictionary.* Berlin: Springer; 2008.
25. Mehrotra BN, Sinha S, Pant P, Seth R. Compendium of Indian medicinal plants: 1960–1969. Vol. 1. In: Rastogi RP, editor. *Drug research perspectives.* New Delhi: Central Drug Research Institute and Publications & Information Directorate; 1990.
26. Anbalagan K, Sadique J. *Withania somnifera* (Ashwagandha), a rejuvenating herbal drug which controls alpha-2-macroglobulin synthesis during inflammation. *Pharm Biol.* 2008;46:177–183.
27. Dhuley JN. Effect of ashwagandha on lipid peroxidation in stress-induced animals. *J Ethnopharmacol.* 1998;60(2):173–178.
28. Bone K. *Clinical applications of Ayurvedic and Chinese herbs: Monographs for the Western Herbal.* Queensland (Australia): Phytotherapy Press; 1996. p. 7–41.
29. Kulkarni SK, George B. Anticonvulsant action of *Withania somnifera* root extract against PTZ-induced kindling in mice. *Phytother Res.* 1996;10:305–312.
30. Kulkarni SK, Sharma AC, Verma AK, Ticku MK. GABA receptor-mediated anticonvulsant action of *Withania somnifera* root extract. *Indian Drugs.* 1993;30:305–312.
31. Ziauddin M, Phansalkar N, Patki P, Diwanay S, Patwardhan B. Studies on the immunomodulatory effects of ashwagandha. *J Ethnopharmacol.* 1996;50(2):69–76.
32. Kuttan G. Use of *Withania somnifera* Dunal as an adjuvant during radiation therapy. *Indian J Exp Biol.* 1996;34(9):854–856.
33. Bhattacharya SK, Bhattacharya D, Sairam K, Ghosal S. Effect of *Withania somnifera* glycowithanolides on a rat model of tardive dyskinesia. *Phytomedicine.* 2002;9(2):167–170.
34. Ven Murthy MR, Ranjekar PK, Ramassamy C, Deshpande M. Scientific basis for the use of Indian Ayurvedic medicinal plants in the treatment of neurodegenerative disorders: ashwagandha. *CNS Agents Med Chem.* 2010;10(3):238–246.
35. Durg S, Dhadde SB, Vandal R, Shivakumar BS, Charan CS. *Withania somnifera* in neurobehavioural disorders induced by brain oxidative stress in rodents: a systematic review and meta-analysis. *J Pharm Pharmacol.* 2015;67(7):879–899.
36. Wollen KA. Alzheimer's disease: the pros and cons of pharmaceutical, nutritional, botanical, and stimulatory therapies, with a discussion of treatment strategies from the perspective of patients and practitioners. *Altern Med Rev.* 2010;15(3):223–244.
37. Singh RH, Narsimhamurthy K, Singh G. Neuronutrient impact of Ayurvedic Rasayana therapy in brain aging. *Biogerontology.* 2008;9(6):369–374.
38. Kuboyama T, Tohda C, Komatsu K. Effects of ashwagandha roots on neurodegenerative diseases. *Biol Pharm Bull.* 2014;37(6):892–897.
39. Konar A, Shah N, Singh R, Saxena N, Kaul SC, Wadhwa R, Thakur MK. Protective role of Ashwagandha leaf extract and its component withanone on scopolamine-induced changes in the brain and brain-derived cells. *PLoS One.* 2011;6(11):e27265.
40. Kumar P, Singh R, Nazmi A, Lakhanpal D, Kataria H, Kaur G. Glioprotective effects of ashwagandha leaf extract against lead-induced toxicity. *Biomed Res Int.* 2014;2014:182029.

41. Bhattacharya SK, Satyan KS. Experimental methods for evaluation of psychotropic agents in rodents: I—anti-anxiety agents. *Indian J Exp Biol.* 1997;35(6):565–575.
42. Parihar MS, Hemnani T. Alzheimer's disease pathogenesis and therapeutic interventions. *J Clin Neurosci.* 2004;11(5):456–467.
43. Jain S, Shukla SD, Sharma K, Bhatnagar M. Neuroprotective effects of *Withania somnifera* in hippocampal sub-regions of female albino rat. *Phytother Res.* 2001;15(6):544–548.
44. Parihar MS, Hemnani T. Phenolic antioxidants attenuate hippocampal neuronal cell damage against kainic acid-induced excitotoxicity. *J Biosci.* 2003;28(1):121–128.
45. Chaudhary G, Sharma U, Jagannathan NR, Gupta YK. Evaluation of *Withania somnifera* in a middle cerebral artery occlusion model of stroke in rats. *Clin Exp Pharmacol Physiol.* 2003;30(5–6):399–404.
46. Raghavan A, Shah ZA. *Withania somnifera* improves ischemic stroke outcomes by attenuating PARP1–AIF-mediated caspase-independent apoptosis. *Mol Neurobiol.* 2015;52(3):1093–1105.
47. Raghavan A, Shah ZA. *Withania somnifera*: a pre-clinical study on neuroregenerative therapy for stroke. *Neural Regen Res.* 2015;10(2):183–185.
48. Mukherjee S, Kumar G, Patnaik R. Withanolide A penetrates the brain via intranasal administration and exerts neuroprotection in cerebral ischemia–reperfusion injury in mice. *Xenobiotica.* 2020;50(8):957–966.
49. Patel P, Julien JP, Kriz J. Early-stage treatment with withaferin A reduces levels of misfolded superoxide dismutase 1 and extends lifespan in a mouse model of amyotrophic lateral sclerosis. *Neurotherapeutics.* 2015;12(1):217–233.
50. Bhattacharya SK, Bhattacharya A, Sairam K, Ghosal S. Anxiolytic–antidepressant activity of *Withania somnifera* glycowithanolides. *Phytomedicine.* 2000;7(6):463–469.
51. Ramanathan M, Balaji B, Antony J. Behavioral and neurochemical evaluation of Perment, an herbal formulation, in chronic unpredictable mild stress-induced depressive model. *Indian J Exp Biol.* 2011;49:269–275.
52. Kulkarni SK, Dhir A. *Withania somnifera*: an Indian ginseng. *Prog Neuropsychopharmacol Biol Psychiatry.* 2008;32(5):1093–1105.
53. Singh N, Bhalla M, de Jager P, Gilca M. An overview on ashwagandha: a Rasayana (rejuvenator) of Ayurveda. *Afr J Tradit Complement Altern Med.* 2011;8(5 Suppl):208–213.
54. Tiwari S, Atluri V, Kaushik A, Yndart A, Nair M. Alzheimer's disease: pathogenesis, diagnostics, and therapeutics. *Int J Nanomedicine.* 2019;14:5541–5554.
55. Ahmad M, Saleem S, Ahmad AS, Ansari MA, Yousuf S, Hoda MN, Islam F. Neuroprotective effects of *Withania somnifera* on 6-hydroxydopamine induced Parkinsonism in rats. *Hum Exp Toxicol.* 2005;24(3):137–147.
56. Mehta V, Chander H, Munshi A. Mechanisms of anti-tumor activity of *Withania somnifera* (Ashwagandha). *Nutr Cancer.* 2021;73(6):914–926.
57. Surya Ulhas R, Malaviya A. In silico validation of novel therapeutic activities of withaferin A using molecular docking and dynamics studies. *J Biomol Struct Dyn.* 2023;41(11):5045–5056.
58. Dar NJ, Hamid A, Ahmad M. Pharmacologic overview of *Withania somnifera*, the Indian ginseng. *Cell Mol Life Sci.* 2015;72(23):4445–4460.
59. Nayak S, Nayak S, Panda BK, Das S. A clinical study on management of stress in type 2 diabetes mellitus (Madhumeha) with ashwagandha (*Withania somnifera*). *Ayushdhara.* 2015;2(6):413–417.
60. Shree P, Mishra P, Selvaraj C, Singh SK, Chaube R, Garg N, Tripathi YB. Targeting COVID-19 (SARS-CoV-2) main protease through active phytochemicals of ayurvedic medicinal plants - *Withania somnifera* (Ashwagandha), *Tinospora cordifolia* (Giloy) and *Ocimum sanctum* (Tulsi) - a molecular docking study. *J Biomol Struct Dyn.* 2022;40(1):190–203.

**How to cite this article:** Kumar D, Kaur P, Sharma N, Gaba P, Sharma RK, Sharma S. Ashwagandha (*Withania somnifera*): Herbal Remedies in Neurodegenerative Disorders. *Pharm Res Bull.* 2026;5(1):124–132.