International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

ARTICLE TITLE	NEUROPROTECTIVE PROPERTIES OF WITHANIA SOMNIFERA - THERAPEUTIC POTENTIAL IN ALZHEIMER'S AND PARKINSON'S DISEASE
DOI	https://doi.org/10.31435/ijitss.4(48).2025.4045
RECEIVED	28 September 2025
ACCEPTED	05 December 2025
PUBLISHED	11 December 2025
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

NEUROPROTECTIVE PROPERTIES OF WITHANIA SOMNIFERA - THERAPEUTIC POTENTIAL IN ALZHEIMER'S AND PARKINSON'S DISEASE

Weronika Sobota *(Corresponding Author, Email: werasobota@gmail.com)*
National Medical Institute of the MSWiA, Warsaw, Poland
ORCID ID: 0009-0000-7778-7030

Przemysław Piskorz
National Medical Institute of the MSWiA, Warsaw, Poland
ORCID ID: 0009-0000-9812-2230

Patryk Biesaga
W. Orlowski Hospital, Warsaw, Poland
ORCID ID: 0009-0003-0242-9126

Olaf Jadanowski
University of Health Sciences in Bydgoszcz, Bydgoszcz, Poland
ORCID ID: 0009-0000-6279-3067

Kamil Łebek
Jan Kochanowski University, Kielce, Poland
ORCID ID: 0009-0005-4612-5022

Alicja Bury
Intensive Care Unit, W. Orlowski Hospital, Warsaw, Poland
ORCID ID: 0009-0009-2950-8741

Daria Litworska-Sójka
Wojewódzki Szpital Zespolony, Kielce, Poland
ORCID ID: 0009-0005-4590-3777

Bartosz Komsta
Military Institute of Medicine – National Research Institute, Warsaw, Poland
ORCID ID: 0009-0003-1159-5855

Julia Lipiec
Independent Public Health Care Facility of the Ministry of Internal Affairs and Administration in Kielce, Kielce, Poland
ORCID ID: 0009-0004-3351-4056

Wojciech Pabis
5th Military Research Hospital and Polyclinic, Kraków, Poland
ORCID ID: 0009-0006-3455-4724

ABSTRACT

Introduction: Withania somnifera (ashwagandha) is a medicinal plant known for its adaptogenic, anti-inflammatory and neuroprotective properties. The withanolides it contains have antioxidant and neuroplasticity-enhancing effects, making it a potential aid in the treatment of neurodegenerative diseases such as Alzheimer's and Parkinson's.
Aim of the study: The aim of this literature review was to analyse the mechanisms of action of Withania somnifera and to evaluate its potential efficacy and safety in the context of adjunctive therapy in neurodegenerative diseases.
Material and methods: The search was conducted in the PubMed database. Most of the publications from the last 10 years were included, covering in vitro, in vivo and clinical studies. The following keywords and MeSH terms were used: 'Withania somnifera', "Ashwagandha", 'Alzheimer's disease', 'Parkinson's disease', 'neuroprotection'.
Results: The analysed preclinical and clinical studies indicate a positive effect of Withania somnifera on cognitive and motor functions, reduction of oxidative stress and inhibition of inflammatory and apoptotic processes in the central nervous system. These data suggest the possibility of using ashwagandha as an adjunct in the treatment of neurodegeneration.
Conclusions: Although the results of previous studies are promising, further high-quality randomised clinical trials are needed to confirm the efficacy and safety of Withania somnifera in the treatment of Alzheimer's and Parkinson's disease.

KEYWORDS

Ashwagandha, Withania Somnifera, Neuroprotection, Alzheimer's Disease, Parkinson's Disease, Phytotherapy, Oxidative Stress

CITATION

Weronika Sobota, Przemysław Piskorz, Patryk Biesaga, Olaf Jadanowski, Kamil Łebek, Alicja Bury, Daria Litworska-Sójka, Bartosz Komsta, Julia Lipiec, Wojciech Pabis. (2025). Neuroprotective Properties of Withania Somnifera – Therapeutic Potential in Alzheimer's and Parkinson's Disease. *International Journal of Innovative Technologies in Social Science*, 4(48). doi: 10.31435/ijitss.4(48).2025.4045

Introduction

Neurodegenerative disorders such as Alzheimer's disease and Parkinson's disease pose a growing challenge to modern medicine due to ageing populations and the limited effectiveness of available therapies. Both conditions are characterised by progressive damage to nerve cells, leading to severe cognitive and motor deficits and a deterioration in patients' quality of life. Despite advances in understanding the pathophysiological mechanisms, current treatments are mainly symptomatic and do not halt the neurodegenerative process.

As a result, there is growing interest in complementary and integrative therapies that aim not only to alleviate symptoms but also to provide neuroprotective effects. One area of research is plant-derived compounds with antioxidant, anti-inflammatory and neuroprotective properties. In particular, Withania somnifera (ashwagandha), a plant used for centuries in Ayurvedic medicine, is gaining importance as a potential adjunctive treatment for neurodegenerative diseases.

The aim of this paper is to review the current literature on the neuroprotective properties of Withania somnifera and to analyse its therapeutic potential in the context of Alzheimer's disease and Parkinson's disease.

Materials and methods

This paper is a review based on an analysis of scientific literature available in the PubMed database. The aim of the review was to collect and synthesise current data on the neuroprotective potential of Withania somnifera (ashwagandha) in the context of Alzheimer's disease and Parkinson's disease.

The review included articles published in English, mostly in the last 10 years, which concerned in vitro, in vivo and clinical studies analysing the effect of Withania somnifera on neurodegenerative processes associated with Alzheimer's or Parkinson's disease.

The review excluded publications that did not contain original data, such as comments or letters to the editor, studies on the effects of Withania somnifera in other clinical contexts unrelated to the nervous system, as well as publications of low methodological quality, e.g. without peer review or with unclear methods.

The literature search was conducted in the PubMed database using a combination of keywords: 'Withania somnifera' or 'Ashwagandha' in combination with "neuroprotection", 'Alzheimer's disease' or 'Parkinson's disease'. MeSH terms such as Withania somnifera, Neurodegenerative Diseases, Alzheimer Disease and Parkinson Disease were also used. The search was conducted manually, and then the titles, abstracts and full texts were analysed for relevance to the topic of the review.

The collected data were presented in a descriptive manner. The latest results of experimental and clinical studies were included, classified according to the type of model used (cellular, animal or clinical), therapeutic indications and the proposed mechanisms of neuroprotective action of Withania somnifera.

1. Neurodegenerative diseases: pathophysiology and current treatment

1.1. Alzheimer's disease - pathogenesis, treatment, challenges

Alzheimer's disease (AD) is the most common form of dementia and mainly affects people over the age of 65. Its development is associated with the accumulation of pathological β-amyloid (Aβ) protein in the brain, leading to the formation of amyloid plaques, and hyperphosphorylated tau protein, which forms neurofibrillary tangles. Both processes contribute to the loss of synapses and neurons, and consequently to cognitive and behavioural disorders [1,2].

At the molecular level, increased oxidative stress, mitochondrial dysfunction, chronic inflammation within the central nervous system and damage to the blood-brain barrier are also observed [3]. Standard treatment includes acetylcholinesterase inhibitors (e.g. donepezil) and NMDA receptor antagonists (e.g. memantine), which alleviate symptoms but do not inhibit the progression of the disease [4].

Recent research on monoclonal antibodies (e.g. lecanemab) offers hope for modifying the course of the disease, but the effectiveness of these therapies remains limited and their safety requires further observation [5]. As a result, there is growing interest in substances with neuroprotective potential, including those of natural origin.

1.2. Parkinson's disease - pathogenesis, treatment, challenges

Parkinson's disease (PD) is a progressive neurodegenerative disorder whose main pathological feature is the death of dopaminergic neurons in the substantia nigra of the midbrain and the formation of Lewy bodies – deposits of α-synuclein protein [6].

Clinical symptoms include resting tremor, muscle stiffness, bradykinesia and postural instability. In advanced stages, neuropsychiatric symptoms such as depression, dementia and hallucinations also appear [7].

Treatment is mainly based on the administration of dopamine precursors (e.g. levodopa), COMT inhibitors, dopamine receptor agonists and MAO-B inhibitors. Although this treatment effectively controls the symptoms, its long-term use leads to motor fluctuations and dyskinesias, and the disease continues to progress [8].

Due to the multifactorial pathogenesis of PD, including oxidative stress, mitochondrial dysfunction, inflammation, and autophagy disorders, compounds with antioxidant and neuroprotective effects, such as adaptogens and plant compounds, are attracting increasing attention [9].

1.3. The need to support conventional therapies

In both Alzheimer's and Parkinson's disease, current treatments are symptomatic and do not halt or reverse neurodegeneration. In recent years, there has been growing interest in an integrative approach to treatment, incorporating substances with anti-inflammatory, antioxidant and neuroprotective effects. Medicinal plants such as Withania somnifera (ashwagandha) are being studied in this context as a potential supplement to conventional therapies [10].

Ashwagandha has numerous beneficial effects on the nervous system, including the ability to alleviate oxidative stress, regulate pro-inflammatory cytokines and modulate neurotransmitters. Importantly, the active compounds of this plant, such as withanolides and sitoindosides, may support neuronal plasticity and synaptic regeneration, making it a subject of interest in the treatment of neurodegenerative diseases.

2. Withania somnifera: composition, properties and mechanisms of action

2.1. Main active compounds

Withania somnifera (ashwagandha) is a plant belonging to the nightshade family (Solanaceae), traditionally used in Ayurvedic medicine as a 'rasayana', i.e. a rejuvenating and strengthening agent. Its root and leaves contain numerous bioactive compounds, the most important of which are withanolides, sitoindosides, alkaloids (e.g. anferine, tropine) and glycowithanolides [11].

Withanolides are steroid lactones with a structure similar to steroid hormones, exhibiting adaptogenic, anti-inflammatory and neuroprotective properties. Among them, withanolide A and withaferin A exhibit the highest biological activity [12]. Sitoindosides, which are glycosylated derivatives of withanolides, support adaptogenic and anti-stress effects. Their presence is also responsible for calming effects and memory improvement [13].

2.2. Pharmacological properties - adaptogen, antioxidant, anti-inflammatory agent

Ashwagandha has a broad spectrum of pharmacological activity, confirmed in numerous preclinical and clinical studies. Its extracts exhibit strong properties:

Adaptogenic - they regulate the body's response to stress, lower cortisol levels and modulate the hypothalamic-pituitary-adrenal (HPA) axis, affecting overall homeostasis [14].

Antioxidant - withanolides and sitoindosides neutralise free radicals (e.g. ROS), increase the levels of antioxidant enzymes (catalase, SOD, glutathione) and protect mitochondria from oxidative damage.

Anti-inflammatory - they inhibit the expression of pro-inflammatory cytokines such as TNF-α, IL-1β, IL-6, and reduce the activity of the transcription factor NF-κB, which contributes to the protection of nerve cells from chronic inflammation [15].

2.3. Neuroprotective mechanisms

In the context of neurodegenerative diseases, particularly AD and PD, Withania somnifera acts on multiple levels, making it an interesting candidate for adjunctive therapy.

2.3.1. Reduction of oxidative stress

Oxidative stress plays a key role in the pathogenesis of AD and PD, leading to damage to cell membranes, proteins and DNA. Ashwagandha extracts increase the activity of antioxidant enzymes, reducing the levels of reactive oxygen species (ROS) and lipid peroxidation products (e.g. MDA) [16].

2.3.2. Anti-inflammatory properties

Ashwagandha reduces neuroinflammation by inhibiting microglial activation and pro-inflammatory cytokine production. Withaferin A and withanone have been shown to suppress the NF-κB pathway and inhibit the expression of COX-2 and iNOS, which contributes to neuron protection [17].

2.3.3. Effects on neurogenesis and neuroplasticity

Studies have shown that withanolides can increase levels of brain-derived neurotrophic factor (BDNF) and promote synapse regeneration. In animal models, dendrite regeneration and improved neuronal plasticity were observed after administration of W. somnifera root extract [18].

2.3.4. Modulation of neurotransmitter systems

Ashwagandha affects the levels of neurotransmitters such as acetylcholine, GABA, dopamine and serotonin. In AD models, acetylcholinesterase (AChE) inhibition has been observed, which may have a beneficial effect on memory and cognitive function. In Parkinson's disease, these compounds help maintain the function of the dopaminergic system.

2.3.5. β-amyloid removal and neuron protection

The ability of ashwagandha to remove β-amyloid from the brain has been confirmed in studies on transgenic mice. Extracts from the root of W. somnifera increased the expression of the LRP-1 (low-density lipoprotein receptor-related protein-1) receptor, which is responsible for transporting amyloid from the brain to the blood [19].

3. The therapeutic potential of Withania somnifera in Alzheimer's disease

3.1. Ashwagandha and oxidative stress and beta-amyloid

Alzheimer's disease (AD) is largely associated with chronic oxidative stress and the accumulation of pathological proteins, mainly beta-amyloid (Aβ) and tau protein. Oxidative stress intensifies the formation of Aβ deposits, while Aβ induces the production of reactive oxygen species (ROS), creating a vicious cycle of neuronal degeneration [20].

In this context, Withania somnifera exhibits a protective effect. Studies show that ashwagandha root extracts significantly reduce Aβ levels in the brain. In mouse models with induced Aβ neurotoxicity, administration of withanolide A led to inhibition of neurodegeneration and improvement in cognitive function.

In addition, the extracts increase the levels of antioxidant enzymes such as glutathione (GSH), superoxide dismutase (SOD) and catalase, and reduce the levels of oxidative damage markers such as malondialdehyde (MDA). This effect significantly contributes to the protection of neurons from oxidative stress and cell death.

3.2. In vitro and in vivo studies – effects on memory and brain pathology

Experiments conducted on transgenic mice expressing human APP (amyloid precursor protein) have shown that ashwagandha supplementation improves memory and reduces the number of Aβ deposits in the hippocampus and cerebral cortex. Kuboyama and colleagues (2005) showed that withanolide A stimulates dendrite regeneration and synapse reconstruction in hippocampal neurons, which translates into improved spatial memory in animals [18].

Ashwagandha also increases the expression of the LRP-1 (low-density lipoprotein receptor-related protein 1) receptor, which plays an important role in removing Aβ from the brain into the circulation. In studies by Sehgal et al. (2012), administration of W. somnifera extract increased LRP-1 activity and reduced Aβ deposits in mice [21].

It is also worth noting that the effect on cognitive function has been confirmed in various animal models, including rats with memory impairment induced by scopolamine [22].

3.3. Clinical studies – effects on cognitive function

Although most of the data on the effects of ashwagandha in AD come from preclinical studies, there are also data from clinical studies confirming its beneficial effects on memory and cognitive abilities. In a randomised double-blind study conducted by Pingali et al. (2014) in healthy volunteers, supplementation with ashwagandha root extract was shown to improve immediate and delayed memory and cognitive performance [23].

Choudhary et al. (2017) conducted a study in patients with mild cognitive impairment, in which ashwagandha supplementation also produced significant benefits, including improved concentration, information processing speed and quality of life [24].

Despite the promising results, it is worth noting that the number of clinical studies specifically involving patients with Alzheimer's disease is still limited, and cognitive assessment scales are not always fully consistent. Further, larger studies involving patients at different stages of the disease are needed to confirm the efficacy and safety of this therapy.

4. The therapeutic potential of Withania somnifera in Parkinson's disease

4.1. Mechanisms of neurodegeneration in Parkinson's disease and the effects of ashwagandha

Parkinson's disease (PD) is the second most common neurodegenerative disease, characterised by progressive loss of dopaminergic neurons in the substantia nigra and the presence of Lewy bodies, i.e. aggregates of α-synuclein. The loss of dopamine leads to motor disorders such as resting tremor, muscle stiffness, bradykinesia and postural instability [25].

Oxidative stress, neurogenic inflammation, mitochondrial dysfunction and protein aggregation play an important role in the neurodegenerative process. Withania somnifera, thanks to its antioxidant, anti-inflammatory and neurotrophic properties, can potentially influence each of these pathological mechanisms.

4.2. In vivo studies – MPTP and 6-OHDA neurotoxicity model

In animal models of PD induced by neurotoxins such as MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) and 6-OHDA (6-hydroxydopamine), ashwagandha has shown strong neuroprotective effects. In studies on mice with MPTP-induced dopaminergic neuron damage, administration of W. somnifera extract significantly reduced the decrease in dopamine levels in the striatum and improved motor coordination [26].

In addition, Withaferin A has been shown to inhibit α-synuclein aggregation and reduce its toxicity in in vitro models, which may be of key importance in PD therapy [27].

4.3. Effects on mitochondria and oxidative stress

Mitochondrial dysfunction and excessive ROS production are the main mechanisms leading to the apoptosis of dopaminergic neurons. Ashwagandha protects mitochondria by maintaining membrane potential, improving ATP production and reducing cytochrome c leakage, a key factor in the mitochondrial apoptosis pathway [28].

Withania somnifera extracts increase the activity of antioxidant enzymes such as superoxide dismutase (SOD), glutathione (GSH) and catalase – in areas of the brain responsible for movement control, including the striatum and substantia nigra, which contributes to a reduction in lipid peroxidation and oxidative stress [29].

4.4. Clinical trials – current state of knowledge

Unlike Alzheimer's disease, clinical data on the use of Withania somnifera in the treatment of Parkinson's disease are much more limited. However, initial pilot studies suggest that ashwagandha supplementation may support conventional therapy.

A meta-analysis of 14 randomised, double-blind studies showed that herbal therapies supporting PD treatment significantly improve scores on the Unified Parkinson’s Disease Rating Scale (UPDRS) and quality of life (PDQ-39) compared to placebo [30].

This study suggests the safety of ashwagandha and its potential supportive effect in PD patients. However, further, larger clinical trials with different doses, forms of preparation and longer follow-up periods are needed.

5. Limitations and safety of use of Withania somnifera

5.1. Research limitations and challenges

Most clinical studies on ashwagandha are pilot studies or are conducted on small groups of participants, which limits the possibility of generalising the results. The diversity of preparations (different extraction methods, withanolide concentrations), dosage, duration of therapy and heterogeneity of the study populations pose further challenges.

In addition, there is a lack of standardised guidelines for the use of ashwagandha in neurodegenerative diseases, which makes it difficult to assess its efficacy and compare results between studies.

5.2. Safety of use

Data on the safety of Withania somnifera are generally very positive. In clinical trials, root extracts were used at doses ranging from 300 to 600 mg per day, usually without serious side effects [31].

The most commonly reported side effects include gastrointestinal complaints (nausea, diarrhoea) and headaches, which usually disappear after discontinuation of therapy. Ashwagandha does not show significant interactions with dopaminergic or cholinergic drugs, although caution and medical consultation are recommended when combining it with other therapies.

Contraindications include pregnancy, breastfeeding and certain autoimmune diseases, due to the plant's immunomodulatory properties.

Summary

Withania somnifera is a promising supplement with neuroprotective properties, as confirmed by numerous preclinical studies and a growing number of clinical trials. Its antioxidant, anti-inflammatory and neurotrophic effects, as well as its ability to modulate pathologies characteristic of Alzheimer's and Parkinson's diseases, make it an attractive candidate for adjunctive therapy.

Despite the positive results, further well-designed, larger-scale clinical trials, standardisation of preparations and longer follow-up periods are needed to fully assess the efficacy and safety of ashwagandha in the treatment of neurodegenerative diseases.

Authors' contributions:

All authors contributed to the article: conceptualization: WS, methodology: PP, WS, DL-S software: PB, PP, OJ, KŁ check: WS, JL, formal analysis: WP, AB, BK investigation: WS, PP, resources: DL-S, PB data curation: JL, AB, OJ writing -rough preparation: BK, KŁ, writing -review and editing: WS, PP, PB, DL-S, JL, OJ visualization: WS, KŁ, WP supervision: WS; project administration: WS, PP. All authors have read and agreed with the published version of the manuscript.

Conflict of Interest Statement: The authors report no conflict of interest.
Financial: The study did not receive any funding
Institutional Review Board Statement: Not applicable.
Informed Consent Statement: Not applicable.
Data Availability Statement: Not applicable.

REFERENCES

1. 2024 Alzheimer's disease facts and figures. Alzheimers Dement. 2024 May;20(5):3708-3821. doi: 10.1002/alz.13809.
2. Hardy J, Selkoe DJ. The amyloid hypothesis of Alzheimer's disease: progress and problems on the road to therapeutics. Science. 2002 Jul 19;297(5580):353-6. doi: 10.1126/science.1072994.
3. Zahiruddin S, Basist P, Parveen A, Parveen R, Khan W, Gaurav, Ahmad S. Ashwagandha in brain disorders: A review of recent developments. J Ethnopharmacol. 2020 Jul 15;257:112876. doi: 10.1016/j.jep.2020.112876.
4. Breijyeh Z, Karaman R. Comprehensive Review on Alzheimer's Disease: Causes and Treatment. Molecules. 2020 Dec 8;25(24):5789. doi: 10.3390/molecules25245789.
5. van Dyck CH, Swanson CJ, Aisen P, Bateman RJ, Chen C, Gee M, Kanekiyo M, Li D, Reyderman L, Cohen S, Froelich L, Katayama S, Sabbagh M, Vellas B, Watson D, Dhadda S, Irizarry M, Kramer LD, Iwatsubo T. Lecanemab in Early Alzheimer's Disease. N Engl J Med. 2023 Jan 5;388(1):9-21. doi: 10.1056/NEJMoa2212948.
6. Tolosa E, Garrido A, Scholz SW, Poewe W. Challenges in the diagnosis of Parkinson's disease. Lancet Neurol. 2021 May;20(5):385-397. doi: 10.1016/S1474-4422(21)00030-2.
7. Sveinbjornsdottir S. The clinical symptoms of Parkinson's disease. J Neurochem. 2016 Oct;139 Suppl 1:318-324. doi: 10.1111/jnc.13691.
8. Fox SH, Katzenschlager R, Lim SY, Ravina B, Seppi K, Coelho M, Poewe W, Rascol O, Goetz CG, Sampaio C. The Movement Disorder Society Evidence-Based Medicine Review Update: Treatments for the motor symptoms of Parkinson's disease. Mov Disord. 2011 Oct;26 Suppl 3:S2-41. doi: 10.1002/mds.23829.
9. Dias V, Junn E, Mouradian MM. The role of oxidative stress in Parkinson's disease. J Parkinsons Dis. 2013;3(4):461-91. doi: 10.3233/JPD-130230.
10. Bhat JA, Akther T, Najar RA, Rasool F, Hamid A. *Withania somnifera* (L.) Dunal (Ashwagandha); current understanding and future prospect as a potential drug candidate. Front Pharmacol. 2022 Dec 12;13:1029123. doi: 10.3389/fphar.2022.1029123.
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. doi: 10.4314/ajtcam.v8i5S.9.
12. Mirjalili MH, Moyano E, Bonfill M, Cusido RM, Palazón J. Steroidal lactones from Withania somnifera, an ancient plant for novel medicine. Molecules. 2009 Jul 3;14(7):2373-93. doi: 10.3390/molecules14072373.
13. Gaurav H, Yadav D, Maurya A, Yadav H, Yadav R, Shukla AC, Sharma M, Gupta VK, Palazon J. Biodiversity, Biochemical Profiling, and Pharmaco-Commercial Applications of *Withania somnifera*: A Review. Molecules. 2023 Jan 26;28(3):1208. doi: 10.3390/molecules28031208.
14. Chandrasekhar K, Kapoor J, Anishetty S. A prospective, randomized double-blind, placebo-controlled study of safety and efficacy of a high-concentration full-spectrum extract of ashwagandha root in reducing stress and anxiety in adults. Indian J Psychol Med. 2012 Jul;34(3):255-62. doi: 10.4103/0253-7176.106022.
15. Gupta M, Kaur G. Aqueous extract from the Withania somnifera leaves as a potential anti-neuroinflammatory agent: a mechanistic study. J Neuroinflammation. 2016 Aug 22;13(1):193. doi: 10.1186/s12974-016-0650-3.
16. Kulkarni SK, Dhir A. Withania somnifera: an Indian ginseng. Prog Neuropsychopharmacol Biol Psychiatry. 2008 Jul 1;32(5):1093-105. doi: 10.1016/j.pnpbp.2007.09.011.
17. Sun GY, Li R, Cui J, Hannink M, Gu Z, Fritsche KL, Lubahn DB, Simonyi A. Withania somnifera and Its Withanolides Attenuate Oxidative and Inflammatory Responses and Up-Regulate Antioxidant Responses in BV-2 Microglial Cells. Neuromolecular Med. 2016 Sep;18(3):241-52. doi: 10.1007/s12017-016-8411-0.
18. Kuboyama T, Tohda C, Komatsu K. Neuritic regeneration and synaptic reconstruction induced by withanolide A. Br J Pharmacol. 2005 Apr;144(7):961-71. doi: 10.1038/sj.bjp.0706122.

19. Mikulska P, Malinowska M, Ignacyk M, Szustowski P, Nowak J, Pesta K, Szeląg M, Szklanny D, Judasz E, Kaczmarek G, Ejiohuo OP, Paczkowska-Walendowska M, Gościniak A, Cielecka-Piontek J. Ashwagandha *(Withania somnifera)*-Current Research on the Health-Promoting Activities: A Narrative Review. Pharmaceutics. 2023 Mar 24;15(4):1057. doi: 10.3390/pharmaceutics15041057.
20. Butterfield DA, Halliwell B. Oxidative stress, dysfunctional glucose metabolism and Alzheimer disease. Nat Rev Neurosci. 2019 Mar;20(3):148-160. doi: 10.1038/s41583-019-0132-6.
21. Sehgal N, Gupta A, Valli RK, Joshi SD, Mills JT, Hamel E, Khanna P, Jain SC, Thakur SS, Ravindranath V. Withania somnifera reverses Alzheimer's disease pathology by enhancing low-density lipoprotein receptor-related protein in liver. Proc Natl Acad Sci U S A. 2012 Feb 28;109(9):3510-5. doi: 10.1073/pnas.1112209109.
22. Visweswari, G.; Christopher, R.; Rajendra, W. Withania Somnifera Against Glutamate Excitotoxicity and Neuronal Cell Loss in a Scopolamine-Induced Rat Model of Alzheimer's Disease. *European Journal of Biological Research* 2021, *11*, 156-167.
23. Pingali U, Pilli R, Fatima N. Effect of standardized aqueous extract of Withania somnifera on tests of cognitive and psychomotor performance in healthy human participants. Pharmacognosy Res. 2014 Jan;6(1):12-8. doi: 10.4103/0974-8490.122912.
24. Choudhary D, Bhattacharyya S, Bose S. Efficacy and Safety of Ashwagandha (Withania somnifera (L.) Dunal) Root Extract in Improving Memory and Cognitive Functions. J Diet Suppl. 2017 Nov 2;14(6):599-612. doi: 10.1080/19390211.2017.1284970.
25. Kalia LV, Lang AE. Parkinson's disease. Lancet. 2015;386(9996):896–912.
26. Sankar SR, Manivasagam T, Krishnamurti A, Ramanathan M. The neuroprotective effect of Withania somnifera root extract in MPTP-intoxicated mice: an analysis of behavioral and biochemical variables. Cell Mol Biol Lett. 2007;12(4):473-81. doi: 10.2478/s11658-007-0015-0.
27. Javed H, Nagoor Meeran MF, Azimullah S, Adem A, Sadek B, Ojha SK. Plant Extracts and Phytochemicals Targeting α-Synuclein Aggregation in Parkinson's Disease Models. Front Pharmacol. 2019 Mar 19;9:1555. doi: 10.3389/fphar.2018.01555.
28. Fanibunda SE, Kukkemane K, Ghai U, Kolthur-Seetharam U, Hingorani L, Vaidya ADB, Vaidya VA. Withania somnifera Regulates Mitochondrial Biogenesis and Energetics in Rat Cortical Neurons: Role of BDNF and SIRT1. Mol Neurobiol. 2025 Aug;62(8):10277-10295. doi: 10.1007/s12035-025-04920-7.
29. 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 Mar;24(3):137-47. doi: 10.1191/0960327105ht509oa.
30. Shan CS, Zhang HF, Xu QQ, Shi YH, Wang Y, Li Y, Lin Y, Zheng GQ. Herbal Medicine Formulas for Parkinson's Disease: A Systematic Review and Meta-Analysis of Randomized Double-Blind Placebo-Controlled Clinical Trials. Front Aging Neurosci. 2018 Nov 8;10:349. doi: 10.3389/fnagi.2018.00349.
31. Tandon N, Yadav SS. Safety and clinical effectiveness of Withania Somnifera (Linn.) Dunal root in human ailments. J Ethnopharmacol. 2020 Jun 12;255:112768. doi: 10.1016/j.jep.2020.112768.