Scientific Reports of Medicinal Plants Used for the Prevention and Treatment of Neurodegenerative diseases

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Abstract
Neurodegenerative diseases are incurable and debilitating conditions that result in progressive degeneration / death of nerve cells or neurons in the human brain. Alzheimer's disease and Parkinson's disease are the major Neurodegenerative diseases, which are characterized by progressive loss (and even death) of structure and function of neurons, and have created great burden to the individual and the society. Treatment of these diseases with prolonged administration of synthetic drugs will lead to severe side effects. Therefore herbal treatment is being preferred over conventional treatments. Much attention and so scope is drawn towards herbal remedy of many Neurodegenerative diseases. The present review puts together research on various medicinal plants that have shown promise in reversing the Alzheimer's and Parkinson’s pathology and highlights importance of phytochemicals of medicinal herbs on neuroprotective function and their mechanism of action.

1 Introduction

Neurons are the building blocks of the nervous system which includes the brain and spinal cord. Neurons normally don’t reproduce or replace themselves, so when they become damaged or die they cannot be replaced by the body. Neurodegenerative diseases are incurable and debilitating conditions that result in progressive degeneration / death of nerve cells or neurons in the human brain. This causes problems with movement (called ataxias), or mental functioning (called dementias).

The actual cause of various neurodegenerative diseases still remains a mystery in healthcare. Some of the commonly studied environmental factors causes for neurodegenerative diseases are protein degradation, oxidative stress, inflammation, environmental factor, mitochondrial defects, familial history, and abnormal protein accumulation in neuron. Ageing is considered as one of the major problem in neurodegenerative diseases. Examples of neurodegenerative diseases include Parkinson’s, Alzheimer’s, and Huntington’s disease, Motor neurone diseases, Spinocerebellar ataxia (SCA), Spinal muscular atrophy (SMA).

2 Alzheimer's disease (AD)

On the other hand, Alzheimer's disease is associated with progressive memory loss, as well as judgment and decision making impairments, according to statistics collected by Guttmacher et al (2003). It is an age-related neurodegenerative disorder characterized by memory deficits. No cure for Alzheimer's exists, and the drugs currently available to treat the disease have limited effectiveness. AD is named after German physician Aloes Alzheimer, who first described it in 1906 and primarily affects the elderly population of over 65 years of age, and is estimated to account for 50 - 60% of the dementia cases. Symptoms typically appear after age 60, and some early-onset forms of the disease are linked to a specific genetic defect. Early disease shows a loss of short-term memory, inability to learn new information, mood swings, difficulty in finding words, forgetting names, and losing items. Frustration, hostility, and irritability are common emotional features exhibited by patients with AD. In severe cases, patients become totally incontinent, memory is completely lost, and sense of time and place disappears.
Several studies have revealed that natural antioxidants, such as vitamin E, vitamin C, and beta-carotene, may help in scavenging free radicals generated during the initiation and progression of these diseases. The loss of memory is considered to be the result of a shortage of the nerve transmitter acetylcholine. It is possible to increase the level of this transmitter in the brain by inhibiting the activity of the enzyme acetylcholinesterase, which splits or breaks down the transmitter substance. Drugs that inhibit the breakdown of the messenger or transmitter acetylcholine delay the development of the disease.

3 Parkinson’s disease (PD)

Parkinson’s disease is a progressive disorder which causes slow motion and rigidity in the body. The clinical manifestation includes bradykinesia (having difficulties in initiating movement), hypokinesia, rigidity, rest tremor and non-motor features including depression, psychosis autonomic dysfunciton. Other symptoms includes co-ordinate movements, shuffling gait, fixed facial expression, lack of blinking, and micrographia, autonomic dysfunction, cognitive, psychiatric changes, sensory symptoms, Seborrhea and Muscle atrophy.

It is characterized by neuronal loss in the substantia nigra and other brain regions, also associated with the formation of intracellular protein inclusions known as Lewy bodies. The loss of dopaminergic neurons, leads to the reduction of dopamine being released into the striatum. Mitochondrial dysfunction and oxidative insult are considered to be the key culprit. The current therapy available for PD primarily relies on Levodopa that offers the potential of slowing down disease progression to some extent but includes lot of side effects. In 1817 James Parkinson first described as paralysis agitans or shaking palsy, the term “Parkinson’s disease” being coined later by Jean-Martin Charcot.

The neurochemical events associated with Parkinson’s disease include increased levels of free radicals, oxidative stress, inflammation, mitochondrial dysfunction, and α-synuclein aggregation. Additionally, increased concentration of redox active metals such as iron and copper, reduced glutathione levels, and increased lipid peroxidation have also been reported. Recent studies have suggested that oxidative stress (OS), produces apoptosis which results in mitochondrial defects, neuroinflammation may also play important roles in its pathogenesis. Various agents as 6-Hydroxydopamine (6-OHDA), 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine, Rotenone a neurotoxin commonly and many more are used in models of PD, induces selective catecholaminergic cell death, mediated by reactive oxygen species (ROS) and mitochondrial defects.

4 Medicinal plants used for the treatment of Alzheimer’s and Parkinson’s disease

A number of scientific researches have been carried out on medicinal herbs. Herbal medicine offers several options to modify the progress and symptoms of Alzheimer’s and Parkinson’s disease. Herbal medicines viz. Ginseng, Ashwagandha, Bacopa monnieri, Ginkgo biloba, Valeriana officinalis, Nardostachys jatamansi, Withania somnifera, Centella asiatica have compounds such as flavonoids, celastrol, trehalose, lycopene, sesamol, resveratrol, and curcumin has gained a lot of interest for their therapeutic potential. Phytocompounds from medicinal plants play a major part in maintaining the brain’s chemical balance by acting upon the function of receptors for the major inhibitory neurotransmitters. Herbs have anti-inflammatory and antioxidant activities that may be used in the treatment of Alzheimer’s and Parkinson’s disease.

The present review puts together research on various Ayurvedic medicinal plants that have shown promise in reversing the Alzheimer’s and Parkinson’s pathology. The present review puts together research on various medicinal plants that have shown promise in reversing the Alzheimer’s and Parkinson’s pathology and highlights importance of phytochemicals of medicinal herbs on neuroprotective function and their mechanism of action. The report summarizes information concerning the phytochemical and biological activities of these various plants in order to provide sufficient baseline information that could be used in drug discovery campaigns and development processes, thereby providing new functional leads for Alzheimer’s and Parkinson’s disease. Table 1 and 2 summarizes some of the commonly used Medicinal plants for their neuroprotective effect.

Table 1: Medicinal plants used in Alzheimer’s disease

<table>
<thead>
<tr>
<th>Plants</th>
<th>Phytoconstituents</th>
<th>Mode of action</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acorus Calamus</strong></td>
<td>α- and β-asarone, 15-methoxyipinolsolidic acid, isopimarane diterpene, ent-isopimara-15-en-3 alpha, 8 alpha-diol diterpenes, lambertianic acid, 15-dien-18-oic acid</td>
<td>Inhibits the acetylcholinesterase</td>
<td>Lannert et al.</td>
</tr>
<tr>
<td><strong>Bacopa</strong></td>
<td>Bacoside A, Bacoside, Betulinic acid, D-</td>
<td>It inhibited cholinergic degeneration and</td>
<td>Chatterji et</td>
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<tr>
<td><strong>monniera</strong></td>
<td>Mannitol, Stigmastanol, b-Sitosterol, Stigmasterol</td>
<td>displayed a cognition-enhancing effect in a rat model. 14Uabundit et al.</td>
<td></td>
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<tr>
<td><strong>Centella asiatica</strong></td>
<td>Asiatic acid &amp; asiaticoside</td>
<td>It inhibits beta-amyloid cell death in vitro, suggesting a possible role for gotu kola in the treatment and prevention of AD and beta-amyloid toxicity 15Cervenka et al., 16Dhanasekaran et al.</td>
<td></td>
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<tr>
<td><strong>Celastrus paniculatus</strong></td>
<td>Triacylglycerol, free fatty acids, diacylglycerol, esterified sterols, &amp; monoacylglycerol</td>
<td>The aqueous extracts have dose-dependent cholinergic activity, thereby improving memory performance 17Ramadan et al., 18Rocha et al.</td>
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<tr>
<td><strong>Collinsonia canadensis</strong></td>
<td>Carvacol &amp; thymol</td>
<td>Prevent the breakdown of acetylcholine</td>
<td>19<a href="http://www.livestrong.com">http://www.livestrong.com</a>.</td>
</tr>
<tr>
<td><strong>Commiphora mukul</strong></td>
<td>Terpenes, sesquiterpenoids, cuminic aldehyde, eugenol, and the ketone steroids Z- and E-guggulsterone, guggulsterols I, II, &amp; III</td>
<td>Decreased neuronal cholesterol levels, in turn, inhibit the beta-amyloid-forming amyloidogenic pathway, possibly by anti-acetylcholine esterase activity 20Urizar et al., 21Vestergaard et al.</td>
<td></td>
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<tr>
<td><strong>Convolvulus pluricaulis</strong></td>
<td>Convoline, convoline, convolvine, confoline, convosine, kamperol &amp; steroids phytosterol</td>
<td>Hippocampal regions associated with the learning and memory functions showed a dose-dependent increase in acetylcholine esterase activity in the CA1 and CA3 area 22Sharma et al.</td>
<td></td>
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<tr>
<td><strong>Crocus sativus</strong></td>
<td>Genticis, gallic acids, lycopene, picrocrocin, safranal, crocin, zeaxanthin, α- and β-carotenes</td>
<td>Extract used in the treatment of mild-to-moderate Alzheimer's disease 23Kyriakides et al., 24Akhondzadeh et al.</td>
<td></td>
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<tr>
<td><strong>Curcuma longa</strong></td>
<td>Essential oils, curcumin, polyphenol</td>
<td>It involves inhibition of articular NF-B, a transcription factor activated in vascular endothelium and synovial cells in RA joints 25Funk et al., 26Henrotin et al.</td>
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<tr>
<td><strong>Chamomilla recutita</strong></td>
<td>Bisabol oxide A, alpha-bisabolol, bisabol oxide B, cis-enzyme-bicycloether, bisabolon oxide A, chamazulene, spathulenol &amp; (E)-beta-farnesene</td>
<td>Extracts of plant might inhibit morphine dependence and withdrawal possibly by increasing cyclic adenosine monophosphate (c-AMP) levels 27Ross et al., 28Tayel et al.</td>
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<tr>
<td><strong>Evodia rutaecarpa</strong></td>
<td>Rutacearpine, limonin, wuchuyuamide I, evocarpine, taraxerone, methyl coumarate, caffeine</td>
<td>It inhibits prostaglandin and/or COX-2 production, using one or more indolequinazoline alkaloids 29Kobayashi et al., 30Liao et al.</td>
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<tr>
<td><strong>Galanthus nivalis</strong></td>
<td>Galanthamine, an isoquinoline alkaloid</td>
<td>Specific inhibitor of the Acetylcholinesterase enzyme and to potentiate cholinergic nicotinic neurotransmission by allosterically modulating the nicotinic acetylcholine receptors 31Takay et al., et al.</td>
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<tr>
<td><strong>Ginkgo biloba</strong></td>
<td>Terpene triactones, ginkgolides A, B, C, J and bilobalide, biflavones, proanthocyanidins, alklyphenols, polyphenols</td>
<td>It acts to varying degrees as scavengers for free radicals, which considered the mediators of the excessive lipid peroxidation, decline of membrane fluidity, and cell damage observed in AD 32Teris et al., 33Le Bars et al.</td>
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<tr>
<td><strong>Glycyrrhiza glabra</strong></td>
<td>Glycyrrhizin, glycyrrhizic, glycyrrhetic acid &amp; two molecules of glucuronic acid</td>
<td>Inhibition of viral binding to cell membranes and replication, as well as interference with cellular signal transduction 34Siracusa et al., 35Isbrucker et al.</td>
<td></td>
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<tr>
<td><strong>Huperzia serrata</strong></td>
<td>Lycocepherramine H, serratidine, obscurumine A, 11α-O-acetylycypodine,</td>
<td>Extract serves as a powerful inhibitor to an enzyme called acetylcholinesterase (AChE) 36Rafii et al.</td>
<td></td>
</tr>
<tr>
<td>Plant Name</td>
<td>Key Constituents</td>
<td>Activity</td>
<td>Reference(s)</td>
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<tr>
<td>Lepidium meyenii</td>
<td>Acyclic keto, alkaloids, amino acids, arginine, histidine, phenylalanine, threonine, tyrosine, anthocyanines, glucotropaeolin</td>
<td>It exerts its antioxidant and AChE inhibitory activities</td>
<td>Fu et al. (2019)</td>
</tr>
<tr>
<td>Magnolia Officinalis</td>
<td>Magnolol &amp; honokiol</td>
<td>Inhibits the memory impairment induced by scopolamine through the inhibition of acetylcholinesterase</td>
<td>Fu et al. (2019)</td>
</tr>
<tr>
<td>Melissa officinalis</td>
<td>Caffeic acid, luteolin-7-O-glucoside, isoquercitrin, rhamnocitrin, rosmarinic acid, ferulic acid, methyl carnosolate, hydroxyccinnamic acid</td>
<td>Reduced agitation and Alzheimer's symptoms</td>
<td>Basta et al. (2019)</td>
</tr>
<tr>
<td>Panax ginseng</td>
<td>Ginsenosides, or saponins, 20(S)-protopanaxadiol (PPD) and 20(S)-protopanaxatriol (PPT)</td>
<td>It involves inhibition of generation or aggregation of amyloid beta (Aβ), enhancement of the removal of Aβ from the neurons, interruption of hyperphosphorylation</td>
<td>Tawab et al. (2019), Imbimbo et al. (2019)</td>
</tr>
<tr>
<td>Pinus pinaster</td>
<td>Catechin, taxifolin, procyanidins, catechin, epicatechin units, phenolic acids</td>
<td>Desmethoxyyangonin, one of the six major kavalactones, is a reversible MAO-B inhibitor and is able to increase dopamine levels in the nucleus accumbens</td>
<td>Peng et al. (2019)</td>
</tr>
<tr>
<td>Rosmarinus officinalis</td>
<td>Carnosic acid, rosmarinic acid, camphor, caffeic acid, ursolic acid, betulenic acid, rosmaridphenol &amp; rosmanol</td>
<td>Carnosol and carnosic acid, which have been shown to be powerful inhibitors of lipid peroxidation</td>
<td>Katerinopoulos et al. (2019)</td>
</tr>
<tr>
<td>Rheum glabraicaule</td>
<td>n-hexacosanic acid, palmitic acid, daucosterol, chrysophanol-8-Me ether, citreorosein, chrysophanol 8-O-beta-D-glucopyranoside</td>
<td>Rhapontigenin exerted a dose-dependent protective effect on mitochondrial functioning against amyloid beta (1-42) neurotoxicity</td>
<td>Wei et al. (2019), Misiti et al. (2019)</td>
</tr>
<tr>
<td>Salvia officinalis</td>
<td>Cineole, borneol, thujone, tannic acid, oleic acid, ursolic acid, cornsoline, fumaric acid, chlorogenic acid, caffeic acid, nicotinamide</td>
<td>It possesses powerful antioxidant properties as well as Acetylcholinesterase-inhibiting compounds</td>
<td>Spiridonov et al. (2019), Akhondzadeh et al. (2019)</td>
</tr>
<tr>
<td>Salix alba</td>
<td>Salicin, salicortin, populin, fragilin, tremulacin, Salicyl alcohol, saligenin, salidroside, vanillin, syringin, salicylic acid, caffeic and ferulic acids</td>
<td>Salicin is a nonselective COX-1 and COX-2 inhibitor, effectively acting as an anti-inflammatory by blocking prostaglandin release</td>
<td>Schmid et al. (2019)</td>
</tr>
<tr>
<td>Terminalia chebula</td>
<td>Arjunglucoside I, arjunigenin, chebulosides I and II, chebulinic acid, gallic acid, ethyl gallate, punicalagin</td>
<td>It exert acetylcholinesterase inhibitory and has suggested developing this herb as a potential in the treatment of AD</td>
<td>Ali et al. (2019), Sancheti et al. (2019)</td>
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</tbody>
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Urtica dioica
- Acetylcholine, histamine, 5-hydroxytryptamine, protein, fat, fiber
  - It enhances the cholinergic system in the brain
  - Withanamides has been shown to scavenge free radicals generated during the initiation and progression of AD. Neuronal cell death triggered by amyloid plaques was also blocked by withanamides
  - Kavalali et al.

Withania somnifera
- Withanolides A to Y, dehydrowithanolide R, withasomniferin A, withasomdielon, withasomiferols A to C, withaferin A, & withanolide R
- It also seems to inhibit the synthesis of prostaglandin-E2 (PGE2) and thromboxane B2 (TXB2), it inhibits thromboxane synthetase. It seems to act on serotonin receptors
  - Matsuda et al.
  - Jayaprakasam et al.

Zingiber officinale
- Zingerone, shogaols, gingersols, β-sesquiphellandrene, bisabolene, farnesene, β-phelladrene, cineol, citral
  - Withanamides has been shown to scavenge free radicals generated during the initiation and progression of AD. Neuronal cell death triggered by amyloid plaques was also blocked by withanamides
  - Rhode et al.
  - Aziz et al.

Table 2: Medicinal plants used in Parkinson’s Disease

<table>
<thead>
<tr>
<th>Plants</th>
<th>Phytoconstituents</th>
<th>Mode of action</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allium sativum</td>
<td>S-allylcysteine</td>
<td>Improved locomotor function which correlated with increased dopamine production, alleviated lipid peroxidation and superoxide production, and enhanced superoxide dismutase (SOD) activity</td>
<td>Kim et al., Garcia et al.</td>
</tr>
<tr>
<td>Bacopa monniera</td>
<td>Bacoside A, Bacoside, Betulinic acid, D-Mannitol</td>
<td>Significantly improvements in behavioral activity and restoration of GSH, SOD, and catalase activity levels and reduced lipid peroxidation</td>
<td>Shobana et al.</td>
</tr>
<tr>
<td>Cassia obtusifolia</td>
<td>Rubrofusarin, Isorubrofusarin</td>
<td>Protect against dopaminergic neuronal degeneration in the substantia nigra and striatum of MPTP-induced PD mice models and dopaminergic neurons in vitro. In 6-OHDA induced pc12 cells, CSE supplementation has been demonstrated to mitigate cell damage, attenuate ROS generation and mitochondrial membrane depolarization</td>
<td>Sayre et al.</td>
</tr>
<tr>
<td>Camelia sinesis</td>
<td>Epicatechin-3-gallate</td>
<td>Extract can attenuate DA depletion, iron dyshomeostasis and dopaminergic neuronal survival in the substantia nigra region of the brain. inhibit DA uptake by blocking uptake of the neurotoxin MPP+ and protecting dopaminergic neurons against MPP+ injury. Moreover, it regulates extracellular signaling kinases</td>
<td>Levites et al., Deleu et al., Satoh et al.</td>
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<tr>
<td>Chaenomeles speciosa</td>
<td>Rutin, catechin and epicatechin</td>
<td>Potent DA transport (DAT) inhibitor; attenuates DA uptake by DA transport in Chinese hamster ovary (CHO) cells expressing DAT (D8 cells); maintains cell viability, tyrosine hydroxylase (TH) activity, and behavioral performance in MPTP induced neurotoxic models of PD</td>
<td>An et al.</td>
</tr>
<tr>
<td>Citrus sinensis</td>
<td>Flavonones, flavone glycosides, polymethoxyflavones, naringenin</td>
<td>Mitigate the loss of dopaminergic neurons and tyrosine hydroxylase in the substantia nigra of the 6-OHD rat model</td>
<td>Datla et al.</td>
</tr>
<tr>
<td>Curcuma longa</td>
<td>Curcumin, demethoxycurcumin (DMC), bis-demethoxycurcumin</td>
<td>Protecting dopaminergic neurons against LPS and α-synuclein induced neurotoxicity, mitigating DA loss, which alleviates oxidative stress and limits mitochondrial</td>
<td>Vajragupta et al., Rajeswari et al.</td>
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<tr>
<td>Plant</td>
<td>Compound/Effect</td>
<td>References</td>
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<tr>
<td>Coffea arabica</td>
<td>Caffeine, effective against dopaminergic neuronal loss in MPTP-induced PD mice; causes reversal of motor deficit in PD mice; reduction in apomorphine-induced rotation and enhanced motor function</td>
<td>70Fisone et al., 71Chen et al., 72Aguiar et al.</td>
<td></td>
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<tr>
<td>Ginkgo biloba</td>
<td>Ginkgolides, bilobalides, effective against oxidative stress induced by MPTP in C57BL/6J mice; it recovered striatal DA levels and tyrosine hydroxylase in the striatum and substantia nigra pars compacta; exhibits inhibitory effects on MAO activity on rat mitochondria</td>
<td>73DeFeudis et al., 74Rojas et al., 75White et al.</td>
<td></td>
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<tr>
<td>Lycopersicon esculentum</td>
<td>Lycopene, decreased DA loss in a MPTP-induced PD model</td>
<td>76Suganuma et al.</td>
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<tr>
<td>Morus alba</td>
<td>Polyphenols, anthocyanin, rutin, quercetin and α and γ tocopherol; stabilize the mitochondrial membrane; regulate the expression of Bcl-2, Bax and caspase 3 proteins involved in apoptosis; also alleviate bradykinesia and dopaminergic neuronal damage in the substantia nigra</td>
<td>77Kim et al., 78Zheng et al.</td>
<td></td>
</tr>
<tr>
<td>Mucuna pruriens</td>
<td>Levodopa (L-DOPA), due to modulation of antioxidant enzymes, Bax/Bcl-2 protein ratio, and cleaved caspase-3</td>
<td>79Katzenschlager et al.</td>
<td></td>
</tr>
<tr>
<td>Nigella sativa</td>
<td>Thymoquinone, protect against dopaminergic neuronal deprivation in MPP+ and rotenone induced neurotoxic PD models</td>
<td>80Radad et al.</td>
<td></td>
</tr>
<tr>
<td>Panax ginseng</td>
<td>Ginsenosides, Ginseng, reduce calcium influx, restoring homeostasis, acting as a psychic energizer, free radical generation, protect neurons from mitochondrial dysfunction and glutamate elevation</td>
<td>81Wang et al., 82Kitts et al., 83Wang et al.</td>
<td></td>
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<tr>
<td>Polygala tenuifolia</td>
<td>Xanthones, saponins, oligosaccharide esters, present in extract exerts anti-stress effects through suppression of norepinephrine</td>
<td>84Cheng et al., 85Kawashima et al.</td>
<td></td>
</tr>
<tr>
<td>Polygonum cuspidatum</td>
<td>Resveratrol, emodin, escaped dopaminergic neuronal loss and neurobehavioral defects</td>
<td>86Khan et al.</td>
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<tr>
<td>Pueraria thomsonii</td>
<td>Puerarin, daidzin, daidzein, genistein, showed an inhibitory effect on caspase 3 and caspase 8 activation</td>
<td>87Lin et al., 88Lin et al.</td>
<td></td>
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<tr>
<td>Prunus dulcis</td>
<td>Morin (3,5,7,20,40-pentahydroxyflavone), attenuated behavioral deficits and DA deprivation</td>
<td>89Zhang et al.</td>
<td></td>
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<tr>
<td>Scutellaria baicalensis</td>
<td>Baicalein, baicalin, attenuate iron-induced lipid peroxidation and DA depletion in the substantia nigra. They can also augment GSH levels, hinder α-synuclein aggregation, and reduce iron-induced mitochondrial stress and apoptosis</td>
<td>90Lee et al., 91Cheng et al.</td>
<td></td>
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<tr>
<td>Sesame indicum</td>
<td>Sesamin, sesamol, sesaminol, have a DA enhancing effect in rotenone-induced loss of dopaminergic neurons in mice; modulates the expression of tyrosine hydroxylase, SOD, and catalase, impedes inducible NO synthase protein expression in neuronal cells, and lowers mRNA levels of the potent pro-inflammatory cytokine interleukin-6 (IL-6) in microglial cells</td>
<td>92Lahaie-Collins et al.</td>
<td></td>
</tr>
<tr>
<td>Uncaria rhynchophylla</td>
<td>Rhynchophylline, corynoxine, corynantheine, hirsutine, significant reduction was observed in ROS generation and caspase 3 activity and a remarkable maintenance of cell viability and GSH levels</td>
<td>93Hsieh et al., 94Yamanaka et al.</td>
<td></td>
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<tr>
<td>Plant</td>
<td>Phytoconstituents</td>
<td>Effects</td>
<td>References</td>
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<tr>
<td>Vitis vinifera</td>
<td>Catechins, epicatechins, anthocyanins, and resveratrol</td>
<td>Protect against MPTP induced apoptosis in neuronal cells, with the underpinning mechanism considered to involve the modulation of the expression of pro-apoptotic Bcl-2 gene and antiapoptotic Bax gene</td>
<td>Bournival et al.</td>
</tr>
<tr>
<td>Withania somnifera</td>
<td>Withanone, withaferin, withanolides, withasomidienone, withanolide</td>
<td>Inhibit metastasis and quinone reductase activity, and preferentially affect the cholinergic signal transduction cascade of the cortical and basal forebrain; withanolides are potent suppressors of NF-κB activation; significantly increase catecholamines such as DA, glutathione (GSH), and glutathione (GSH) peroxidase enzyme</td>
<td>RajaSankar et al., RajaSankar et al.</td>
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</table>

5 Conclusion

Medicinal plants served as a platform for ancient Ayurvedic system of medicine. The current review has offered inclusive details of plants used in the treatment of Neurodegenerative diseases special reference to AD and PD. The different phytoconstituents and extracts of various plants demonstrated the protective effect for Neurodegenerative diseases. The existences of bioactive chemicals are mostly responsible for the treatment of AD and PD. However, many other active agents obtained from plants have not been well characterized. It is required to carry out study to assess the mechanism of action of medicinal plants for the treatment of AD and PD.

6 Conflict of interests

None

7 Author’s contributions

SL and JSV collected the data and drafted the manuscript. Both authors have read and approved the final manuscript.

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