INTRODUCTION

Neurodegenerative diseases are characterized by progressive dysfunction and loss of neurons leading to the distinct involvement of functional systems defining clinical presentations [1]. Neurodegeneration is a process involved in both neuropathological conditions and brain aging. It is known that brain pathology in the form of the cerebrovascular and neurodegenerative disease is a leading cause of death all over the world, with an incidence of about 2/1000 and an 8% total death rate [2].

Studies have demonstrated that common pathology of neurodegeneration is deposition of proteins with altered physicochemical properties in the human brain. These pathological conformers are called as misfolded proteins such as accumulation and aggregation of amyloid-β (Aβ) in AD [3], α-synuclein in PD, huntingtin protein in Huntington’s disease (HD), and TDP-43 in frontotemporal dementia (FTD) and amyotrophic lateral sclerosis (ALS) [4]. In addition, it has been recognized that protein elimination pathways, like the ubiquitin-proteasome system and the autophagy-lysosome pathway, clear response proteins and chaperones have a high impact on the pathogenesis [5].

Damage of neurons also contribute to progressive long-term neurodegenerative processes, for example, the role of L-glutamate (or L-aspartate) mediated acute excitotoxicity in cerebral ischemia or status epilepticus is well known. Microglia-mediated neuroinflammation is also one of the most striking hallmarks of various neurodegenerative diseases, including PD, AD, and ALS [6].

The presently available drugs for the treatment of AD are symptomatic only and do not alter the course or progression of the underlying disease and produce adverse reactions in patients thereby having limited scope for the treatment of patients with Alzheimer’s syndrome. Research is also expanding at other substances and treatments that prevent the formation of beta-amyloid plaques, nerve growth factor to keep neurons healthy such as statins, antioxidants (vitamins) and folic acid, anti-inflammatory drugs [7]. The past decade has also witnessed an intense interest in herbal medicines that have long-term health-promoting qualities. Herbal remedies for neurodegenerative disease is becoming more popular in the recent years as they show the possibility to slow down the brain’s degeneration. The benefits derived from using herbal treatments have been very promising as they are not only as effective as prescription drugs but also have fewer side effects [8].

Search criteria

This review included articles from 2003 to 2017 that were found in various electronic databases: PubMed, Science Direct, Scopus, Web of Science, Scirus, and Google Scholar by using the search words: neurodegenerative diseases, neuroprotection, medicinal plants, antioxidant, Alzheimer’s disease, Parkinsonism, dementia, amyloid-β, acetylcholinesterase. Only current articles that reported the effects of medicinal plants on neurodegenerative diseases were included in our study. Then, a comparison of the related vb mechanisms and evaluation of pharmacological effects in the treatment of disease was done.

Herbal neuroprotective agents and their mechanistic pathway

Neuroprotective agents refer to substances that are capable of preserving brain function and structure by reducing and preventing oxidative stress, mitochondrial dysfunction, inflammation, various forms of neurotoxicity (e.g. excitotoxicity), and protein deficiencies. Specific examples of things that can cause neurodegeneration include: traumatic brain injuries, drug abuse, pharmaceutical medications, schizophrenia, strokes, and dementia but the most common cause of neurodegeneration is oxidative stress and to prevent the effects of any neurodegeneration, considering neuroprotective agents may be beneficial for long-term brain health. Administration of a neuroprotective agent may help minimize the...
effects of chronic conditions that could: kill brain cells, decrease brain volume, and lead to long-term functional impairment. There are more than 120 traditional medicines that are being used for the therapy of CNS disorders in Asian countries [10]. In the Indian system of medicine the following medicinal plants have shown promising activity in neuro-psychopharmacology:

**Acorus calamus**

*Acorus calamus* (Sweet flag) belonging to family Araceae, act as a rejuvenator for the brain and nervous system having beneficial memory enhancing the property, learning performance, and behavior modification. *Acorus calamus* contains a majority of α- and β-asarone, β-asarone has the capability of suppressing beta-amyloid-induced neuronal apoptosis in the hippocampus by reversal down-regulation of Bcl-2, Bcl-w, caspase-3 activation and c-Jun N-terminal kinase (JNK) phosphorylation [11]. Methanolic extracts of the roots containing α-asarone showed inhibitory effect on AChE with an IC50 value of 186μg/ml [12]. *Acorus calamus* has the potential of improving the function of dopaminergic nerve; by increasing striatal extracellular dopamine level and the expression of tyrosine hydroxylase in substantiagria therefore it can play role in PD. *Acorus calamus* also increases DI-1 gene expression in the striatum and therefore acts as neuroprotective for PD [13].

**Allium sativum**

*Allium sativum* (family Amaryllidaceae), commonly known as garlic, is one of the most widely quoted herbs found in the old medicinal literatures mainly for its medicinal potentials in prevention and treatment of cardiovascular and other metabolic diseases, atherosclerosis, hyperlipidemia, thrombosis, hypertension, dementia, cancer and diabetes [14, 15]. Allin (allyl 2-propene thiosulfinate or Diallylthiosulfinate) and allin are the principal bioactive compounds of Allium sativum. S-allyl cysteine (SAC) is the major constituent of aged garlic extract (AGE) which is extensively studied [16, 17]. SAC has both direct and indirect antioxidant activity. Apart from decreasing lipid peroxidation and DNA fragmentation, it also reduces protein oxidation and nitration. In 1-methyl-4-phenyl pyridinium (MPP) and 6-hydroxydopamine (6-OHDA) models of Parkinsonism, SAC protected dopamine levels, oxidative damage and lipid peroxidation. In 3-nitro propionic acid-induced oxidative stress [18, 19]. Allyl-containing sulfides in garlic cause the upregulation of neuroprotective proteins such as mitochondrial uncoupling proteins. Allin also activates transient receptor potential ion channels in the plasma membrane of neurons [20, 21].

**Bacopa monnieri**

*Bacopa monnieri* (Linn), commonly referred to as "Brahmi," from the plant family Scrophulariaceae is a creeping herb found in India and neighbouring tropical countries. Steroidal saponins and bacosides A plant family Scrophulariaceae is a creeping herb found in India and *Bacopa monnieri* increased manganese and copper/zinc superoxide dismutase both learning and memory [22]. Other constituents include bacopa neuronal survival [18, 19]. Allyl -containing sulfides in garlic cause indirectly, activates expression of important genes needed for oxidative damage and lipid peroxidation. In 3-nitro propionic acid [23]. *Bacopa monnieri* saponins D, E and F as well as alkaloids, flavonoids, and phytosterols of neuronal survival [18, 19]. Allyl -containing sulfides in garlic cause indirectly, activates expression of important genes needed for oxidative damage and lipid peroxidation. In 3-nitro propionic acid [23]. *Bacopa monnieri* saponins D, E and F as well as alkaloids, flavonoids, and phytosterols. *Bacopa monnieri* also stimulates a significant decrease in the brain levels of MDA [24]. Bacoside A inhibits lipid peroxidation by modulating the effects of enzymes like Hsp 70 and iNOS. Curcuminoids, polyphenol compounds from turmeric belonging to the Zingiberacea family, is a gold-coloured Spice that has been used as a traditional medicine. Curcumin, the principal constituent of turmeric, has several known neuroprotective actions [33]. In Alzheimer’s disease, it has been shown that curcumin has the ability to bind Aβ peptides, prevent aggregation of new amyloid deposits and promote disaggregation of existing amyloid deposits [34]. Scientific studies also reported that curcumin and its analogues demethoxycurcumin and bis-demethoxycurcumin can protect cells from Aβ-induced oxidative stress [35]. Curcumin has the ability to inhibit Aβ oligomerization and fibril formation, enhance Aβ uptake by macrophages and inhibits the peroxidase activity of A beta-heme complex [36]. Curcuminoids, polyphenol compounds from turmeric attenuate mitochondrial dysfunction induced oxidative stress and inflammatory responses to inflammatory cytokines, COX-2, and iNOS. Curcuminoids also bind to Aβ plaques to inhibit amyloid accumulation and aggregation in the brain [37, 38].

**Celastrus paniculatus**

*Celastrus paniculatus* Wild *Celastrus paniculatus* Wild commonly known as jyotishmati belongs to family Celastraceae. In the traditional system of medicine, it was administered as a powerful brain tonic, appetite stimulant, and emetic [39]. Phytochemical studies show the presence of envoioneate, a sesquiterpene, alkaloids paniculatine A, paniculatine B and wiformine F, celastrine, celapamine, celapangine, celapangine, polyalcohol like malangunin, malkanginnol, malkanginol and paniculatadiol, triterpenoids, sterols such as α-amyrin and β-sitosterol [40, 41]. The scientific studies suggested that *Celastrus paniculatus* water extract protected neuronal cells against glutamate-induced toxicity by modulating glutamate receptor function and showed an improvement in learning and memory. It also stimulates a significant decrease in the brain levels of MDA which is an important marker of oxidative damage, with simultaneous significant increases in levels of glutathione and CAT; two endogenous antioxidants in the brain [42]. Research carried out by Jakka AL also elucidated the neurotrophic potential of flavonoids present in Celastruspaniculatus Wild whole plant methanolic extract (CPPME) and treatment with CPPME demonstrated decrease in Aβ aggregation and enhanced neurotrophic activity and thus, ultimately improving spatial memory formation in scopolamine-induced amnesia [43].

**Coriandrum sativum**

*Coriandrum sativum* L. *Coriandrum sativum* L. commonly known as dhanya belongs to family Apiaceae [44]. Major phytochemical present includes flavonoids like...
quercetin 3-glucoronide; polyphenolics like caffeic acid, protocatechnic acid, and glycin. The flavonoid content of the seeds was reported to be 12.6 quercetin equivalents/kg and the polyphenolic content was reported to be 12.2 gallic acid equivalents/kg [45, 46]. A study showed that the extract of Coriandrum sativum increased enzyme levels of SOD, CAT and in the total protein [58]. A study indicated that essential oils and tocopherol content in seeds decreased cerebral lipid peroxidation (LPO) and calcium levels in the experimental rat. Memory deficits induced by scopolamine and diazepam was also reversed by leaves of Coriandrum sativum [47]. It also decreases reactive changes in brain histology like gliosis, lymphocytic infiltration and cellular edema which assist the protective role in cerebrovascular insufficiency states. The leaves also show antioxidant property having 2, 2-diphenyl-1-pircyhydral (DPPH) radical scavenging activity, lipoxigenase inhibition and phospholipid peroxidation inhibition activity, which may also contribute to its memory enhancement effect [48].

Galanthus nivalis

Galanthus nivalis commonly known as snowdrop belongs to family Amaryllidaceae. The major constituent found in bulbs and flowers of Galanthus nivalis is galanthamine which is a tertiary isoquinoline alkaloid. The neuroprotective effect of galanthamine is associated with the dual action of this alkaloid. The drug is a competitive and selective AChE inhibitor. It is capable of stimulating nicotinic antioxidant, immunomodulatory activity, antimicrobial activity, anti-inflammatory activity, and mitochondrial dysfunction, as well as anti-inflammation [70].

Ginkgo biloba

This species, belonging to the Ginkgoaceae family, is considered as a ‘living fossil’ [51]. The extract contains 24% of flavonoids fraction which is mainly composed of three flavonoids, quercetin, kaempferol and isorhamnetin and 6% of terpenic lactones that include diterpenic lactones-the ginkgolides A, B, C, J and M, and a sesquiterpene tri lactone-the bilobalide [52]. Extract exhibit the neuroprotection by several mechanisms that include inhibition of membrane lipid peroxidation, anti-inflammatory effects, and direct inhibition of amyloid-b aggregation and anti-apoptotic activities. The flavonoid fraction of Ginkgo biloba (G. biloba) extract is responsible for the antioxidant and free-radical scavenging properties and bilobalide can reduce damage caused by global brain ischemia and excitotoxicity-induced neuronal death [53, 54]. G. biloba extract significantly inhibit the AChE activity in the brain that indicates an increase in the basal level of acetylcholine [55]. Flavonoids alter a number of a biological process like their interactions with neuronal and signalling pathways, expression of proteins required for synaptic plasticity and repair, changes in cerebral blood flow, inhibition of neuropathological process in certain brain regions. A study carried out by Dash SK showed that the extract of G. biloba inhibits the production of brain Aβ levels by lowering cholesterol, as free and circulating free cholesterol that affect amyloidogenesis. It may also influence the formation of Aβ fibrils by increasing gene expression of transthyretin that prevent Aβ aggregation in vitro [56].

Glycyrrhiza glabra

Glycyrrhiza glabra (G. glabra) commonly known as Yashtri-madhuh or liquorice, belongs to family Leguminosae. The major flavonoid of G. glabra is Glabridin that possesses multiple pharmacological activities such as antiviral, anticancer, anti-ulcer, anti-diabetic, antioxidant, immunomodulatory activity, antimicrobial activity, anti-inflammatory activity, and anticonvulsant. Liquorice significantly improved learning and memory but the research have indicated that its consumption improves the general intelligence rather than short-term memory [57]. Glabridin significantly decreases the level of MDA and it elevates the level superoxide dismutase and reduced glutathione in a rat model [58]. A study indicated that administration of G. glabra restored the decreased levels of brain enzymes such as glutamate and dopamine and decreased AChE activity [59].

Hypericum perforatum

Hypericum perforatum (H. perforatum), is also known as hypericum or millepertuis is a member of the family Hypericaceae. Although it has a worldwide distribution, it is mainly native to Europe, western Asia, and northern Africa. Hyperoside is the main active component of H. perforatum. Hypericin, Kaempferol, Biapigenin and quercetin are its other constituents [60]. H. perforatum extract has also been reported to protect against enzymatic (NADPH-dependent) and non-enzymatic (Fe²⁺-corrosive dependent) lipid peroxidation in the cerebral cortex [61]. The extract also protects brain cells from glutamate-induced cytotoxicity by reducing glutathione loss, calcium overload and ROS-mediated cell death [62]. H. perforatum ethanolic extract may improve microglial viability by reducing amyloid-beta mediated toxicity in Alzheimer’s disease [63]. Hypericum perforatum inhibits acetylcholinesterase and butyrylcholinesterase [64]. The uptake of H. perforatum in the brain and increases the level of SOD, CAT, GPx. According to these findings, H. perforatum also acts as an antioxidant and have the ability to bind iron ions and have scavenging action for hydroxyl radical [65].

Lycopersicum esculentum

Lycopersicum esculentum is a genus of club mosses, also known as ground pines or creeping cedar, in the family Lycopodiaceae, a family of fern-allies. The leaves contain a single, unbranched vascular strand and are microphylls by definition. The genus Lycopersicum (Lycopodiaceae), which produces a potential therapeutic agent, huperzine A, for the treatment of AD, has been extensively studied in recent years [66]. Huperzine A is an alkaloid extracted from Lycopersicum peruvianum and has been used for centuries to treat fever, inflammation, blood disorders and schizophrenia [67]. It is a highly selective, reversible, and potent AChE inhibitor, and potency of AChE inhibition is similar or superior to that of physostigmine, galanthamine, donepezil and tacrine [68]. The huperzine A is a strong candidate for treatment of AD. Other potentially beneficial effects, as far as AD is concerned, include protection against Aβ-induced oxidative injury and neuronal apoptosis, regulation of nerve growth factor and reduction in glutamate-induced toxicity. Huperzine A caused a significant increase in ACh levels in rat brain [69]. Huperzine A has several protective effects such as regulating amyloid precursor protein metabolism, protecting against Aβ mediated oxidative stress, apoptosis and mitochondrial dysfunction, as well as anti-inflammation [70].

Melissa officinalis

The leaves of Melissa officinalis L. (Lamiaceae), also known as lemon balm, are used in traditional medicine for its nerve calming and spasmolytic effects. The leaves produce calming and soothing effects through GABA, benzodiazepine receptor [71]. Its extracts contain some compounds such as flavonoids such as quercitrin as well as apigenin, luteolin and phenolic acids. These derivatives inhibit enzymes monoamine oxidases (MAO) and AChE, scavenges these free radicals and prevent apoptosis. The inhibition of these enzyme leads to alleviation of depression symptoms [72]. Research also suggests that Melissa officinalis exert protective activities in the PC12 cell line and might protect neurons from oxidative stress [73].

Ocimum sanctum

Ocimum sanctum, also known as ‘Tulsi’ in Hindi and ‘Holy Basil’ in English belongs to family Labiatae. The plant is also reported to contain alkaloids, glycosides, saponins, and tannins, vitamin C, and maleic acid, citric and tartaric acid [74]. A research conducted by Kusindara et al. indicated that an ethanolic extract derived from leaves of Ocimum sanctum may stimulate and restores the expression of choline acetyltransferase in ageing human cerebral microvascular endothelial cells and could provide nerve protection and increased production of ACh may enhance the memory and cognitive ability [75]. Scientific studies reveal that the hydroalcoholic extract of Ocimum sanctum exhibits strong antioxidant activity against DPPH and hydroxyl radicals which may be due to the high amount of polyphenols and flavonoids. It inhibits lipid peroxidation, ROS generation, DNA damage, and membrane depolarization. It also decreases the lactate dehydrogenase leakage and preserved the cellular morphology, reduced superoxide dimutase and catalase enzyme levels thereby preventing neuronal damage [76].

Panax ginseng

Ginseng belongs to the family Araliaceae growing in north-eastern Asia. It is one of the most widely used herbs for boosting energy [77]. Ginseng root is characterized by the presence of ginsenosides.

Ginseng may provide protection against neurodegeneration by multiple mechanisms. The ginsenosides improve performance in a passive avoidance learning paradigm and the neuroprotection was possible, due to its ability to suppress cellular AChE activity and enhancement of cholinergic metabolism [78]. It also produces a dose-dependent reduction in the β-amyloid deposition or glutamate-induced excitotoxicity; thereby prevent apoptosis and neuronal death. In different experimental models against PD it suppresses nitric oxide (NO) production and tissue necrotic factor-alpha (TNF-α) secretion, inhibits the mRNA expressions of inducible nitric oxide synthase (iNOS), TNF-α, interleukin (IL-1B), cyclooxygenase-2 (COX-2) and reduces the generation of ROS [79].

Rosmarinus officinalis

Rosemary commonly known as Salatapatika, belongs to the family Lamiaceae. It contains several essential oils like carvacrol, eugenol, oleanolic acid, thymol, and ursoic acid; antioxidant constituents such as carnosic acid and ferulic acid [80]. Carnosic acid, which was first isolated from the plant by Wenkert et al. has also shown to have neuroprotective effects on cyanide-induced brain damage in cultured rodent and human-induced pluripotent stem cell-derived neurons in vitro and in vivo in various brain areas of a non-Swiss albino mouse model. Lian et al. have also shown that carnosol and rosmarinic essential oils inhibit the adhesion of tumour necrosis factor-α (TNF-α) induced monocytes to endothelial cells and suppress the expression of intercellular adhesion molecule (ICAM-1) at the transcriptional level in vitro. Meng et al. reported the inhibitory effect of rosmarinic diterpenes on α-secretase which is one of the major proteolytic enzymes that process amyloid precursor protein. The mechanism behind its neuroprotective actions involves not only inhibition of AChE and β-amyloid deposits as well as anti-BuChE (butyrylcholinesterase) activity [81]. Apart from above activities, it possesses cytoprotective, anti-apoptotic and anti-inflammatory activities that also add on to its neuroprotective mechanism [82].

Salvia officinalis

Salvia is an important genus in the Lamiaceae family reputed for improving memory and has a long history of use as memory enhancing agents [83, 84]. The Rosmarin acid and carnosic acid are the main active ingredient of S. officinalis having potential pharmacological effects that include anti-inflammatory and antioxidant properties as well as weak AChE inhibitory effect [85, 86]. It inhibits reactive oxygen species formation, lipid peroxidation, DNA fragmentation, caspase-3 activation and tau protein hyperphosphorylation [87]. All these clinical evidence may help to prevent or reduce the symptoms of dementia. One small pilot trial showed that oral administration of S. officinalis essential oil to 11 patients showing mild-to-moderate symptoms of AD significantly improved cognitive function [88].

Terminalia chebula

Terminalia chebula (T. chebula), called the "King of Medicines" in Tibet, belongs to the family Combretaceae and is one of the most important medicinal plants used in medicines of Ayurveda, Siddha, Unani and Homeopathy [89]. It contains compounds such as triterpene caryophyllsides 1, arjunegenin and the chebuloids 1 and 2, tannins, chebulic acid, chebulinic acid, tannic acid, ellagic acid, 2,4 chebuly -β-D-glucopyranose, gallic acid, ethyl gallate, punicaglactinol, terchebin. Some flavonoids like rutin, rutins, and quercetin are also present. A study reported that T. chebula exhibit analgesic activity that is comparable to standard drug dexam (90, 91). T. chebula has good pharmacological activities relevant to dementia therapy. Sancheti et al. extracted 1, 2, 3, 4, 6-penta-D-galloyl-β-D-glucose extracted from T. chebula by chromatographic methods that were reported to be the potent AChE and Butyrylcholinesterase inhibitor. Sultaman et al. showed that ethyl acetate extract with doses of 0.05, 0.005, 0.001, and 0.025 g/ml inhibited AChE by 29.36%, 32.44%, 45.82%, and 62.32%, respectively. Gallic acid derived from T. chebula exerts anti-inflammatory activity via the down-regulation of the nuclear factor-κB (NF-κB) pathway in the development of inflammatory diseases, both in vitro and in vivo [92]. T. chebula also acts as an antioxidant and its activity is comparable with reference radical scavengers such as quercetin, showing 95% activity with inhibitory concentration (IC50) value 2.2 μg/ml [93]. T. chebula fruit extract may also protect neuronal cells against ischemia, reduces NO production and death rate of microglia cells stimulated by lipopolysaccharide [94].

Tinospora cordifolia

Tinospora cordifolia (T. cordifolia) belongs to the family Menispermaceae, is commonly known as gőle. Chemical constituents derived from the plant are alkaloids, steroids, diterpenoid lactones, aliphatics, and glycosides [95]. T. cordifolia possesses a memory enhancing property which is due immune-stimulation and increased synthesis of acetycholine [96]. T. cordifolia exhibits strong free radical scavenging properties against ROS and reactive nitrogen species as studied by electron paramagnetic resonance spectroscopy. It also increases the level of reduced glutathione, expression of the gamma-glutamyl-cysteine ligase and copper-zinc superoxide dismutase genes, which plays a major role in neuronal injury during hypoxia/ischemia [97]. In addition, T. cordifolia significantly decreases the mRNA expressions of iNOS. T. cordifolia also increases the level of dopamine in the brain. Thus, T. cordifolia has shown to prevent the neurogenedengenic changes and enhance cognition, learning and memory [98].

Withania somnifera

Withania somnifera belongs to the family Solanaceae, popularly known as Ashwagandha is considered as the Indian ginseng. The major constituents of Ashwagandha root are two withanolides, withaferin A and withanolide D. Active withaolides of Withania somnifera have a significant antioxidant function, which is accomplished by increasing the activities of SOD, CAT, and GPx. Ashwagandha is also reported as a Nervine tonic that rejuvenates the cells and boosts energy [99]. According to Rajasankara, oral treatment of PD mice with Withania somnifera root extract (0.1 g/kg body weight) for 7 or 28 d elevated dopamine, 3,4-dihydroxy phenyl acetic acid and homovanillic acid levels in the corpus striatum. Furthermore, it was reported that Withania somnifera treatment increased the level of anti-apoptotic (Bcl-2) proteins and decreased the level of the pro-apoptotic (Bax) proteins in the Maneb-Paraquid-induced dopaminergic neurodegeneration model of PD. [100]. Ashwagandha extract has shown to prevent the lipid peroxidation and increase the antioxidant activity by increasing the free-radical scavenging enzymes levels in the brain. Sitoindosides VII-X and withaferin, other constituents isolated from the aqueous methanol extract of roots of Withania somnifera tend to decrease AChE activity in the brain [101].

Zizyphus jujube

Jujube fruits are used in traditional Korean and Chinese medicine to reduce anxiety and strengthen the stomach and gastrointestinal system. Jujube seeds contain large amounts of terpenoid, flavonoid, phenyl glycosides and alkaloid compounds mucilage, malic acid, citric acid, sugar, protein, organic minerals and vitamin C [102]. The herb exerts inhibitory activity against histamine release and AChE and cyclooxygenase 1 and II inhibitory activity. Flavonoids possess anti-oxidant properties [103] Ce-9-octadecenamide (oleamide), a compound extracted from jujube is reported to have high effect on the activation of acetycholine transferase (34.1%) in the in vitro which leads to the increase in acetycholine level and improves mild to moderate cognitive functions, learning and memory, motor coordination and behavioral disorders [104].

CONCLUSION

The management of neurodegenerative diseases remains a challenge in the modern medicine because of their complicated pathogenesis. Protein misfolding and their accumulation inside or outside of neurons is the key pathological feature in several neurodegenerative diseases including Alzheimer’s, Parkinson’s Huntington’s disease. Herbal medicines are regarded as effective and promising sources of potential neuroprotective agents because of their cognitive benefits and more significantly, their mechanisms of action with respect to the fundamental pathophysiology of the diseases. Our review has acknowledged several herbal medicines...
such as such as *Allium sativum*, *Ginkgo biloba*, *panaxanginseng*, *Terminalia chebula* with potential therapeutic effects for neurodegenerative diseases. It is anticipated that the information provided through this review will help the researcher to provide some evidence and conceptual detail of the benefit of a wide range of herbs as neuroprotective agents.

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**AUTHOR CONTRIBUTION**

Both the authors had contributed equally to the review work.

**CONFLICTS OF INTERESTS**

Both the authors had contributed equally to the review work.

**REFERENCES**

All authors have none to declare


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