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REVIEW ON: ROLE ON NATURAL PRODUCT IN NEUROPROTECTION

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ABSTRACT

Natural products—plant-derived phytochemicals, marine metabolites, and dietary components—have attracted substantial interest as potential neuroprotective agents. This review synthesizes current knowledge on the mechanisms by which natural compounds confer neuroprotection, summarizes key preclinical and clinical evidence for representative molecules (polyphenols, flavonoids, alkaloids, terpenoids, fatty acids, and cannabinoids), discusses formulation and translational challenges (bioavailability, blood–brain barrier permeability, standardization), and proposes directions for future research. Emphasis is placed on mechanistic categories (antioxidant, anti-inflammatory, anti-apoptotic, mitochondrial stabilization, autophagy modulation, and synaptic protection) and on strategies to advance promising compounds toward clinical application.

KEYWORDS: neuroprotection, natural products, phytochemicals, polyphenols, blood–brain barrier, neurodegeneration, antioxidant, inflammation.

1. INTRODUCTION

The progressive malfunction and loss of neuronal structure and function that leads to the death of neuronal cells is known as neurodegeneration.[1, 2] The central nervous system (CNS) is affected by a variety of disorders that cause neurodegeneration. The clinical manifestation of both acute and chronic neurodegenerative diseases is determined by the loss of particular neuronal populations associated with functional neural networks. The phrase

"neurodegenerative disease" refers to a broad category of neurological disorders that mainly impact CNS neurons and are characterized by a progressive loss of neurons in the CNS, which impairs certain brain processes (such as memory, mobility, and cognition).[3]

Traditional herbal remedies were known before the advent of modern scientific approaches to healthcare and are still used by the majority of the population today. The development of a wide range of synthetic medicines, now widely used to treat mild colds and coughs, cancer and central nervous system problems, has undoubtedly influenced Natural products are known and employed since ancient ogies of neurodegenerative diseases. The plant natural products (CNS). Written records and the use of medicinal herbs date back 5000 years. When Friedrich Bayer & Co. brought synthetic acetylsalicylic acid, commonly known as aspirin, a safer synthetic form of salicylic acid, a key component of willow bark, to the world in 1897, the strong traditional links between nature and human health began to unravel. Plant-based medicines are among the earliest medicines documented in the history of even the most basic medical system. They are the most commonly used drugs due to their ubiquitous availability and the widespread perception that they have a safer profile than synthetic drugs. Herbal medicines and products are becoming increasingly popular around the world, not only as a caffeine-free alternative, but also as a dietary supplement for a low-calorie diet.[4]

Acute neurodegeneration is a condition in which neurons are rapidly damaged and usually die in response to a sudden insult or traumatic event such as head injury, strokes, traumatic brain injury, cerebral or subarachnoid hemorrhaged, and is-chemic brain damage.[5] Meanwhile, chronic neurodegeneration is a chronic state in which neurons in the nervoussystem undergo neurodegenerative process that usually starts slowly and worsen over time with multifactorial causes, resulting in the progressive and irreversible destruction ofspecific neuron populations.[3,6,7] the chronic neurodegenerative diseasesinclude Alzheimer’s disease, Huntington’s disease, Parkinson’s disease, and amyotrophic lateral sclerosis.

Various types of biological mechanisms have been associated with neurodegeneration including oxidative stress, neuroinflammation, excitotoxicity, mitochondrial dysfunction, abnormal protein misfolding and aggregation, and apoptosis.these biological processes have been implicated in the progression and pathogenesis of neurodegenerative diseases. To date, extensive studies haveattempted to elucidate the mechanism and potential therapeutic targets to combat neurodegenerative diseases..erefore, neuroprotection strategies and relative mechanisms work best to prevent or delay the process of neurodegeneration through the

interaction with thepathophysiological change process.It is estimated that medicinal plants.[8,9]

Currently make up a significant part of The US pharmaceutical sector. Much of Our current pharmacology is based on Knowledge we have gained from a long History of using therapeutic plants or The heir bioactive components. Aspirin, Digitalis, morphine and quinine, all Commonly used in allopathic medicine, Were first extracted from herbal Compounds. Later, as chemistry and Biochemistry advanced, as did the Development of isolation, separation, And biosynthetic techniques, many Drugs were separated and evaluated for Bioactivity. Most plant compounds have Been synthesized thanks to advances in Organic biochemistry. However, plants Continue to provide some of the most Important medicines with basic raw Materials, but the production of new Medicines from synthetic sources has Shown tremendous improvements, and Antibiotics are emerging as preemin+ent Therapeutics.[10]

Natural products are known and employed since ancient times for their therapeutic properties. In recent years, biological activities, nutritional values, and potential health and therapeutic benefits of natural products and their bioactive compounds have been intensively explored and investigated. Within the past decades, many studies have reported the protective effect of natural products and its bioactive compounds against various diseases such as cardiovascular, diabetes, reproductive, cancer, and neurode-generative diseases. Natural products have emerged as potential neuroprotective agents for the treatment of neurodegenerative diseases. .is review focused on the therapeutic potential of natural products and their bioactive compounds to exert neuroprotective effects on the pathologies of neurodegenerative diseases.[11]

2. Significance of medicinal herb

The World Health Organization reports that over 75% of people utilize herbs as a kind of traditional medicine. Medicinal plants have the ability to produce medications that are crucial for both preserving and improving the health of people and animals worldwide in difficult situations.

These Ayurvedic herbs have been researched by the Indian herbal industry and are currently used in a variety of herbal formulations. They have also been included in the International Pharmacopoeia through a study in ethano-pharmacology and traditional medicine, which

includes in-depth research in the areas of pharmaceuticals, photochemistry, pharmacology, and clinical therapy. Consequently, the development of natural medicine research has moved from herbal stores to drug research labs, leading to impressive research in a number of areas of pharmaceutical research that draw from

Ayurvedic literature, contributing to India's herbal industry's 20% yearly growth.[12,13]

3. Neurodegeneration and Neurodegenerative Diseases

Mechanisms and Potential Therapeutic Target

The hallmark of neurodegenerative diseases, which include Parkinson's disease, Alzheimer's disease, Huntington's disease, and amyotrophic lateral sclerosis, is the progressive and targeted loss of cells in particular vulnerable CNS neuronal populations.[14] Alzheimer's disease is a chronic, age-related, progressive neurological illness that causes behavioral abnormalities as well as memory and cognitive deficits.

Two primary neuropathological features define it

(i) The development and accumulation of extracellular amyloid-beta (Aβ)

(ii) The buildup of intracellular hyperphosphorylated tau proteins in the brain which are referred to as neurofibrillary tangles. A gradual loss of dopaminergic nigrostriatal neurons causes Parkinson's disease, a chronic and progressive neurodegenerative illness that impairs motor function and manifests as resting tremor, postural abnormalities, bradykinesia, and muscle rigidity. The accumulation of intracellular protein aggregates, Lewy bodies, and Lewy neurons—which are primarily made of misfolded and aggregated forms of the presynaptic protein alpha (α)-synuclein— as well as the progressive loss of dopaminergic nigrostriatal neurons are the neuropathological hallmarks of Parkinson's disease.[15,16]

Amyotrophic lateral sclerosis, another progressive neurodegenerative disease, is typified by the gradual degeneration and death of the upper and leading to respiratory failure, paralysis, and death. Although the exact causes of amyotrophic lateral sclerosis are still unknown, a number of factors have been proposed, including environmental factors, mitochondrial dysfunction, oxidative stress, excitotoxicity, autoimmune response, impaired axonal transport, neurofilament aggregation, and genetic factors. A mutation in the gene encoding the copper/zinc superoxide dismutase-1 (SOD1) enzyme is linked to amyotrophic lateral sclerosis.

In contrast, Huntington's illness is typified clinically by aberrant movements, mental disturbances, and cognitive deficiencies, as well as pathologically by increased dopaminergic activity and decreased gamma-aminobutyric acid (GABA) activities in the basal ganglia. In the Huntingtin (HTT) gene, which is found at the short arm of chromosome 4, it is brought on by a trinucleotide repeat expansion of the nucleotides cytosine, adenine, and guanine (a CAG expansion).[17,18]

Numerous biological processes, such as oxidative stress, neuroinflammation, excitotoxicity, mitochondrial dysfunction, and apoptosis, have been linked to the development and pathophysiology of neurodegenerative disorders (see Figure 1). The pathophysiology of common neurodegenerative diseases is influenced by oxidative stress. Cellular damage, impairment of the DNA repair system, and mitochondrial dysfunction are the outcomes of oxidative stress, which is caused by an imbalance in the production of reactive oxygen species (ROS) and an inadequate antioxidant defense capacity. This will hasten the progression of neurodegenerative disease and the neurodegenerative process.[19]

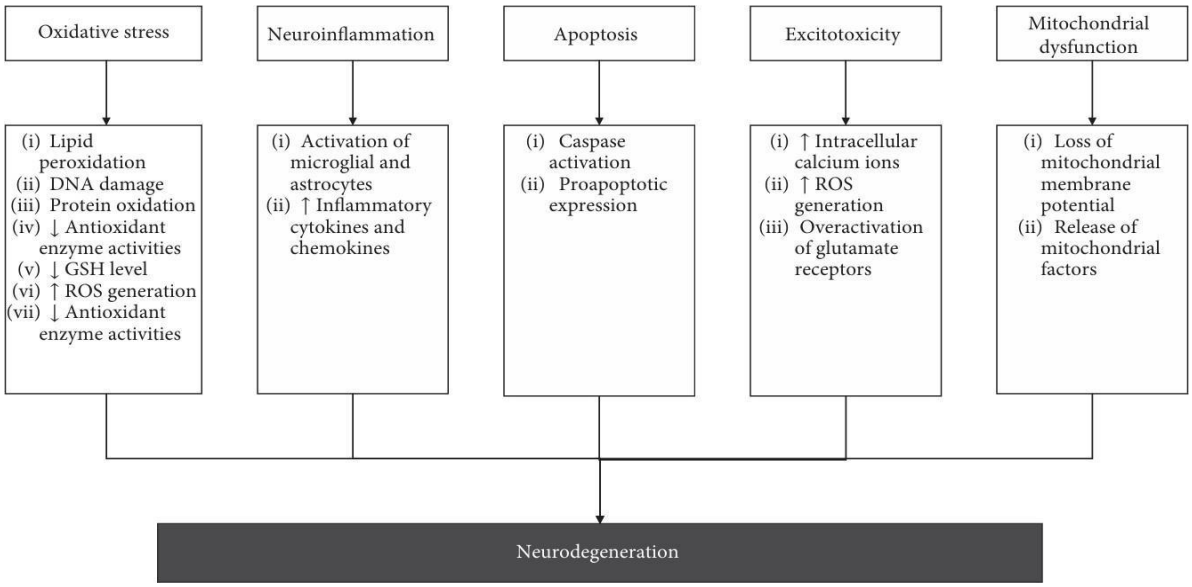

Figure No. 1: Various types of mechanisms have been associated with the neurodegeneration.

Furthermore, the pathogenesis of neurodegenerative disorders has been suggested to involve neuroinflammation. Neurodegeneration has been linked to neuroinflammation, an inflammatory condition that affects the central nervous system's innate and adaptive immune systems. Both the development of the normal brain and neuropathological processes may be influenced by neuroinflammatory pathways. The primary element of innate immune defense

in the central nervous system is microglia. Microglia quickly changed morphologically in response to pathological alterations in the nervous system, and activated microglia release a variety of inflammatory mediators, including cytokines, chemokines, and cytotoxic molecules (cyclooxygenase2 (COX-2), ROS, glutamate, and prostaglandins). These inflammatory mediators will cause astrocytes to initiate a growth factor repair or secondary inflammatory response, and they will also cause neurons to respond for its survival.[20,21]

Neurodegenerative disease etiology may also involve excitotoxicity, another biological mechanism.

It is described as the abnormal process of neuronal death brought on by excitatory amino acids or excitotoxins in the central nervous system activating glutamate receptors excessively or for an extended period of time. Both pathologically elevated glutamate release levels and excitotoxins that bind to glutamate receptors can produce excitotoxicity by facilitating the quick entrance of calcium ions (Ca2+) into the cell. When Ca2+ enters cells, it activates a number of Ca2+- dependent enzymes, such as lipases, phospholipases, endonucleases, xanthine oxidase, protein phosphatases, proteases, protein kinase, and inducible nitric oxide synthase (iNOS). ese enzymes go on to damage cell structures such as components of the cytoskeleton, membrane, and DNA.[22,23] excessive Ca2+ influx could also result in ROS production, mitochondrial dysfunction, oxidative stress, and inflammatory responses. .ese processes ultimately lead to neuronal cell death.

The neurodegenerative process also involves neuronal death. Cell shrinkage, chromatin condensation, DNA fragmentation, and membrane cell death are characteristics of this highly regulated type of cell death. It is an energy-dependent process that needs ATP for signal activation and protein synthesis. Both intrinsic and extrinsic signals can initiate the complex process of apoptosis. The extrinsic pathway includes downstream signaling via a cascade of protein-protein interactions and the activation of death receptors upon ligand binding. In the meantime, the intrinsic route causes either caspasedependent or caspase-independent apoptosis by releasing proapoptotic substances from the mitochondrial intermembrane gap into the cytosol through the mitochondrial permeability transition.[24]

In addition to being the location of oxidative phosphorylation and cellular respiration, mitochondria also help to keep the cytosol's Ca2+ concentration low. The collapse of the

mitochondrial membrane potential and the opening of the mitochondrial permeability transition pores are caused by excessive Ca2+ absorption and ROS production.

The release of mitochondrial factors (cytochrome-c and apoptotic-inducing factor) and mitochondrial uncoupling occur when the outer membrane of the mitochondria ruptures due to the swelling of the mitochondrial matrix caused by the opening of mitochondrial permeability transition pores situated in the cytoplasm through mitochondrial permeability transition holes in the intermembrane space of the mitochondria. In a caspase-dependent mechanism, cytochrome-c forms an apoptosome complex and activates the caspase-3 pathway by binding to apoptotic protease-activating factor-1 and procaspase-9.Apoptotic neuronal death is triggered by caspase activation, which also causes the cleavage of vital cellular substrates such poly (ADP-ribose) polymerase-1 (PARP-1). These changes in mitochondrial function may be a precursor to the loss of neurons. Apoptotic-inducing factor moves to the nucleus and causes chromatin condensation and DNA breakage in caspase-independent mechanisms.[25,26]

Targeting several mechanisms of action is a viable approach for the prevention and treatment of neurodegenerative illnesses as multifarious pathogenic pathways are linked to neurodegeneration. To fight neurodegenerative illnesses, a number of possible therapeutic targets could be investigated (see Figure 2).

Excitotoxicity: One of the most common Pathways for cell death in CNS diseases Is glutamate excitotoxicity. Due to the Lack of selectivity of the glutamate-Binding ion channel, overstimulation Of glutamate receptors, particularly NMDA, increases calcium ion flux. Buffer levels of Ca2+ sequestration With mitochondria are exceeded when Ca2+ accumulates in the neuron with Significant neuronal effects. Ca2+ Is a second messenger that controls A variety of downstream processes. Excess Ca2+ leads to improper control Of these activities, ultimately leading To cell death. Neuroinflammation is an Integral part of many CNS disorders and Is thought to promote Ca2+.

Glutamate antagonists: Glutamate antagonists are the most common treatment for excitotoxicity in CNS disorders and they help prevent or treat it. The purpose of these antagonists is to prevent the buildup of Ca2+ and henexcitotoxicity by inhibiting the binding of glutamate to NMDA receptors. Many glutamate antagonists have been studied as potential

treatments for CNS disorders, however many have proven ineffective or caused severe side effects.[27] Few treatments which have proved potential in the future are the following.

Estrogen: 17-estradiol restricts NMDA receptor and other glutamate receptors that contribute to the regulation of excitotoxicity.

Ginsenoside Rd: ginsenoside Rd, according to the research, inhibits glutamate excitotoxicity. Clinical trials in people who have had an ischemic stroke have revealed that the drug is both effective and noninvasive.[28]

Progestrone: it is well known that progesterone can help avoid subsequent damage in people with traumatic brain injury and stroke.

Simvastatin: simvastatin has been demonstrated to have substantial neuroprotective effects in Parkinson's disease models, including anti-inflammatory effects due to NMDA receptor regulation.[29]

Memantine: memantine reduces NMDA-induced excitotoxicity while Keeping some NMDA signaling as a Noncompetitive low affinity NMDA Antagonist.

Oxidative stress: Increased oxidative stress can be the result of neuroinflammation, which is a known component of cerebral ischemia and numerous neurodegenerative diseases such as Parkinson's disease (PD), Alzheimer's disease (AD), and Amyotrophic lateral sclerosis(ALS). Because of their role in triggering cell death, higher levels of oxidative stress are often addressed in neuroprotective drugs. Oxidative stress can either destroy neurons directly or initiate a chain dysfunction, or activation of glial cells.[30]

Antioxidants: The most common Treatment to reduce oxidative stress Is to increase antioxidant levels. Antioxidants work by ridding the body Of reactive oxygen species, which are the Main cause of neurodegeneration. Some Popular antioxidants that help reduce Oxidative stress are

A. **Acetylcysteine**: In the pathophysiology of a number of neuropsychiatric disorders, Acetylcysteine affects glutamatergic transmission, neuritropins, the Antioxidant glutathione, apoptosis, Mitochondrial activity, and inflammatory Pathways.[31]
B. **Crocin**: it is a saffron derivative discovered to be a powerful antioxidant for the brain.

C. **Estrogen**: the antioxidants 17-estradiol and 17-estradiol have been shown to be beneficial. These drugs have immense potential. The nonestrogenic stereoisomer of 17-estradiol is 17-estradiol.

D. **Oil**: polyunsaturated n-3 fatty acids found in fish oil have been shown To protect against oxidative stress and Mitochondrial dysfunction.

E. **Minocycline**: the semisynthetic tetracycline minocycline can cross the blood-brain barrier. In Alzheimer's disease (AD), Huntington's disease (HD), Parkinson's disease (PD), and Amyotrophic lateral sclerosis, minocycline has been shown to provide neuroprotective benefits in the CNS.[32]

F. **PQQ**: pyrroloquinoline (PQQ) is a neuroprotective antioxidant that works In multiple ways. Resveratrol protects Against oxidative stress by reducing Hydrogen peroxide-induced cytotoxicity As well as cellular reactive oxygen species (ROS) accumulation. It has been shown To protect against a range of neurological Disorders.

Selegiline: in the early stages of Parkinson's disease, it has been found To reduce disease progression and delay The onset of disability by an estimated 9 Months in Parkinson's disease.[33] **Caffeine**: caffeine has been Shown to protect against Parkinson's Disease.

Caffeine promotes cysteine absorption in neurons, leading to glutathione production and neuroprotection. Other neuroprotective treatments There are a variety of neuroprotective therapies available that target distinct pathways of neurodegeneration.

Caspase Inhibitors: caspase inhibitors are widely used and researched for their apoptotic effects.[34] **Trophic Factors**: the role of trophic factors in CNS diseases, particularly ALS, will be investigated. CNTF, VEGF, IGF-1 and BDNF are all potential Neuroprotective trophic factors **Antiprotein aggregation agents**: Neuron cell death is known to be caused By protein aggregation. Antiprotein Aggregation drugs may be helpful. Whether this can be eliminated as The cause of neurodegeneration is Being tested with various treatments. These include sodium, trehalose, Poly-Q binding peptide and sodium 4-phenylbutyrate.[33]

Therapeutic hypothermia: it is being researched as a possibility for neuroprotective therapy in traumatic brain injuries and is intended to reduce intracranial pressure.

Erythropoietin: erythropoietin has been observed to protect nerve cells from Hypoxia-induced toxicity.

Lithium: it inhibits glycogen synthase kinase-3 (GSK3), modulatesneurotropins and growth factors (e.g. BDNF), regulates inflammatory Molecules, modulates neuroprotective Components.

e.g. B–cell lymphoma-2 (Bcl-2) and heat shock protein–70 (HSP-70) and inhibits proapoptotic factors.[35]

4. Neuroprotective Activities of Natural Herbal Products

The therapeutic potential of natural products and their bioactive compounds to exert a neuroprotective effect on the pathologies of neurodegenerative diseases will be discussed in more detail in this section.

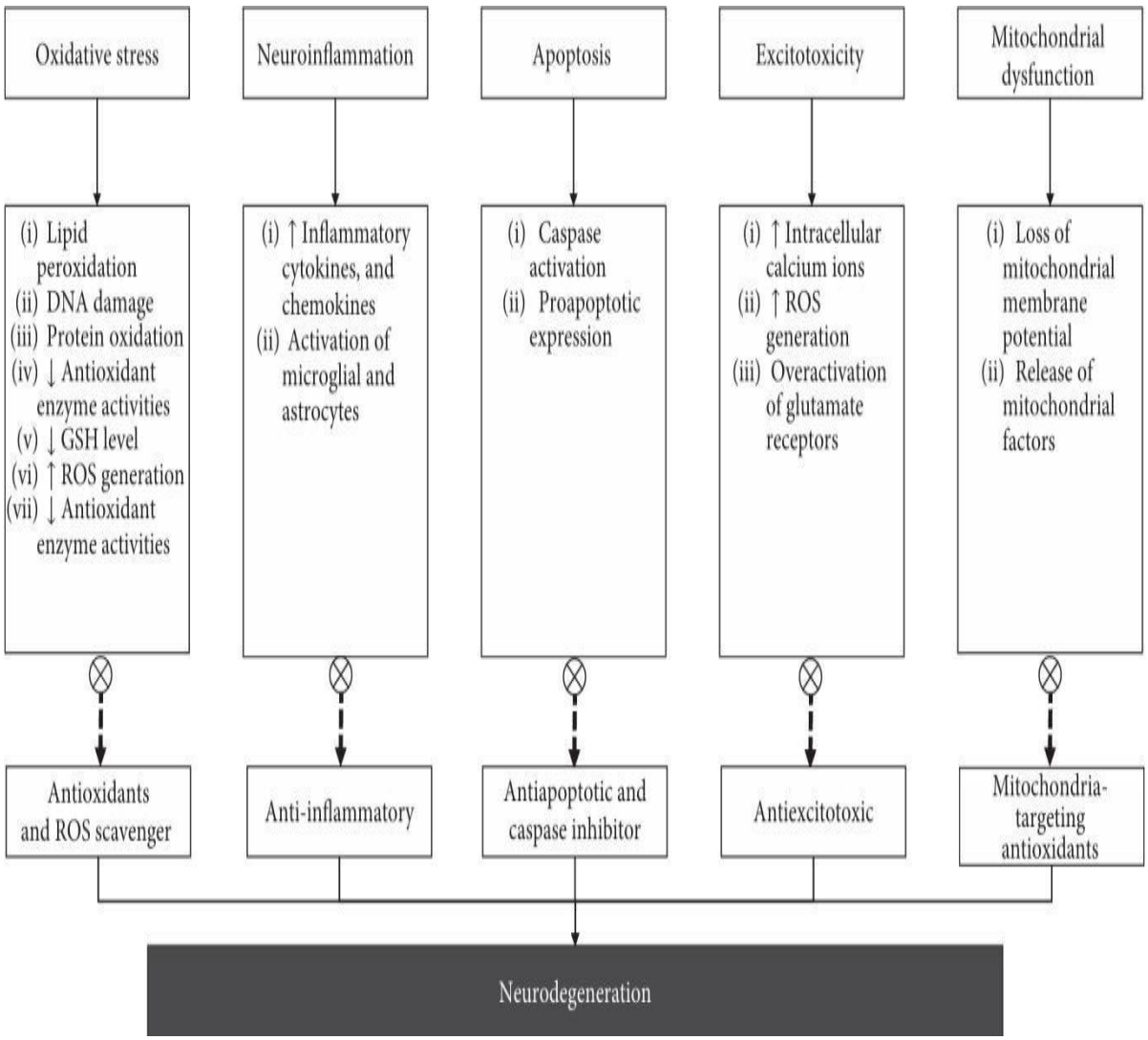

Figure No. 2: The potential therapeutic targets on various mechanisms of neurodegeneration.

1) Hypericum perforatum

Figure No. 1: Hypericum Perforatum.

It is used worldwide as an herbal medicine for depression and related illnesses. A comprehensive analysis found that plant extracts were extremely effective in treating depressive disorders.[36]

2) Ginkgo biloba

Figure No 2: Ginkgo biloba.

Many people believe that G. biloba is a living fossil. One of the world's oldest tree species is the ginkgo biloba. Before being brought to Europe and America as a decorative tree in the 18th century, the tree only made it through Asia's cold period. It has been demonstrated that an extract from the plant's green leaves works well as a vasodilator to treat peripheral circulatory issues. Following observations in both experimental animals and patients, this extract—known as Egb761—was found to be a neuroprotective and CNS.

Function modifying medication. Patients with cerebrovascular disorders or cerebral insufficiency may benefit from its use. Ginkgo is widely used to treat dementia in Europe. It

has eleven flavonoids that support blood flow in the brain and function as antioxidants. Although a number of clinical studies have been revealed to have scientific flaws, ginkgo has been demonstrated to enhance memory, learning, and thought processes. It has shown great efficacy in treating Alzheimer's sufferers. The synergistic effects of ginkgo's constituent parts, which are presentlyunknown, are the basis for its medicinal qualities.[37]

3) Bacopa monniera

Figure No. 3: Brahmi.

Brahmi is another name for Bacopa monniera. It is also referred to as Jalbrahmi or Nirbrahmi in India. It contains bacosides A and B, D-mannitol, saponins, and the alkaloids herpestine and Brahmin. The herb is used to treat headaches, asthma, epilepsy, and foolishness as a diuretic, nerve tonic, and antimicrobial. It is an essential part in the composition of Medhya Rasayana, which promotes memory and learning. Tests utilizing a 50% ethanol extract from a complete plant without roots were the first to demonstrate the impact of this extract.[38]

4) Tinospora cordifolia

Figure No. 4: Tinospora cordifolia.

Numerous studies have demonstrated the substantial immune-stimulating and antistress benefits of Tinospora cordifolia. Tinospora cordifolia is a key component of a restorative

recipe that enhances memory function and is suggested in Ayurvedic literature. Nonetheless, there is experimental proof that Tinospora cordifolia improves memory.[39]

5) Mucuna pruriens

Figure No. 5: Mucuna Pruriens.

In English, this plant is referred to as the Cowage Plant and is also known as Atmagupta. Mucuna Pruriens. Has been acknowledged in Ayurveda as a beneficial medicinal agent for a range of neurological and reproductive disorders. Masabaladfi pacana, an Ayurvedic ingredient that works well for Kampavata, contains mucuna beans. L-dopa was identified in 1937 by Indian researchers Ramaswamy and Damodaran from Mucuna seeds. Whole Mucuna seeds had an L-dopa content of 4.02 percent in HPLC tests.[40]

6) Centellaasiatica

Figure No. 6: Centellaasiatica.

The plant known as Centella asiatica is indigenous to Asia. It is referred to as Mandooki or Indian pennywort. Triterpenoid saponine glycosides, including Asiaticosid, Brahmoside, Brahminoside, and Thankuniside, are present in the medication. Two glucose molecules, a rhamnose, and the aglycone Asiatic acid are produced when the asiaticoside is hydrolyzed. The medication alsocontains amino acids, tannins, sterols, alkaloids, and inorganic salts. The

herb has traditionally been used to cure conditions affecting the skin, nerves, and blood. The leaves are used as a tonic to enhance memory.

7) Galanthus woronowii

Figure No. 7: Galanthus woronowii.

The species Galanthus woronowii Is a member of the Galanthus genus. A snowdrop from the Caucasus is what it is. A pure, unadulterated extract of galanthus is called galantamine. A recent study suggests that galantamine may slow the development of neurodegenerative illnesses. Additionally, it competitively and reversibly inhibits acetylcholine. Galantamine improves the brain's reaction to AChE while decreasing its synthesis. Galantamine was shown to lessen cognitive loss and functional abilities in 653 peoplNe with mild to moderate Alzheimer's disease in one study.[41]

8) Withania somnifera

Ashwagandha, or Withania somnifera, is a Rasayana herb that is utilized in traditional Indian medicine. The seeds and leaves are also used, but mostly for medical purposes. The components with the highest pharmacological activity include steroid lactones and alkaloids. The most prevalent alkaloids are cuscohygrain, tropane, anahygrain, somniferin, anaferine, withamimine, and withaninine, whereas the most prevalent lactones are withanoloids.

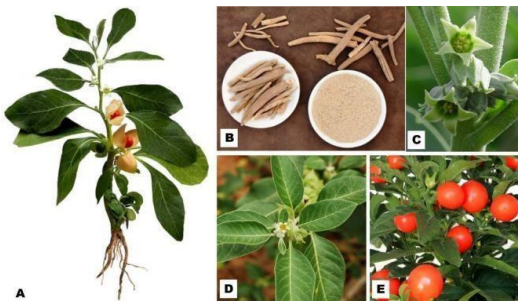

Figure No. 8: Ashwagandha or withania somnifera.

CAT an The total alkaloid content of the root ranges from 0.1% to 0.3%, while higher yields might have been documented. Ashwagandha should be regarded as the most effective herb for all agerelated issues, claims Karnik (1991). There is proof that the medication works well to stop Alzheimer's and senility. Alzheimer's disease impairs memory loss. The medication inhibits the progression of the illness. It has also been found that glycowithanolides (WSG) can reverse the effects of neurotoxic neurodegeneration on central cholinergic markers and cognitive impairment. Ashwagandha was found by Bhattacharya, Ghosal, and Bhattacharya to increase d GPx activity while normalizing SOD and LPO activity.[42]

9) Hemidesmus indicus

Figure No. 9: Hemidesmus indicus.

This plant is indigenous to India and is often referred to as Indian sarsaparilla or anantmool. Studies on mice have demonstrated that it has nootropic (memory-enhancing) potential, and its roots are thought to possess antioxidant properties. It has been demonstrated that jqk n-butanol found in Hemidesmus indicus root extracts enhances memory.[43]

10) Trapa bispinosa

Figure No. 10: Trapa bispinosa.

Since ancient times, people have utilized the medicinal plant Trapabispinosa as a nerve tonic. By lowering lipid peroxidase and activating glutathione peroxidase and catalase, it has been

demonstrated to have neuroprotective effects by reducing oxidative stress brought on by D - galactose.[44]

11) Ocimum sanctum

Figure No. 11: Ocimum Sanctum.

An ethanolic extract of Ocimum sanctum alleviated age-related memory impairments and scopolamine (0.4 mg/kg). The therapy of Alzheimer's disease and other age-related dementias may benefit from this enhancement, which validated cholinergic modulation as a mechanism of action.[45]

12) Clitoria ternatea

Figure No. 12: Clitoria Ternatea.

Well-known medication in Indian Ayurvedic medicine. Using the radial arm maze and the condition avoidance response test, Vyawahare et al. investigated the impact of alcoholic extracts of Clitoriaternatea roots on scopolamine-induced memory impairment.[46]

13) Panax ginseng

Figure No. 13: Panax ginseng.

For thousands of years, people have utilised this plant to treat a range of ailments, including age- related neurodegeneration. It is thought that giving ginseng extract or ginseng-based combination therapies to animals enhances their memory and learning. Dosing decreased amyloid accumulation or glutamate-induced excitotoxicity, which leads to neuronal death and is a primary cause of Alzheimer's disease, according to Wang et al. The herb coriandrum sativum is known to improve blood flow to the brain, memory, and cognitive function. It has anti-free radical and anti- lipid peroxidation properties. It has been found that an aqueous extract of coriander seed increases therapeutic benefits while shielding cerebral cortex pyramidal cells against Alzheimer's disease and neurodegenerative illnesses. Found mostly in western industrialised nations, phenylpropanoids, isoprenoids, and alkaloids can support health benefits through appropriate dietary practices, including defence against chronic degenerative diseases including cancer, heart disease, and neurological diseases.[47]

CONCLUSION

Numerous experimental investigations have demonstrated the neuroprotective potential of natural products and natural bioactive substances against neurodegenerative disorders. To prevent and treat a variety of neurodegenerative disorders without having negative side effects, natural products and their significant bioactive ingredients are essential. Numerous modes of action are crucial for neuroprotection techniques for the prevention and treatment of neurodegenerative illnesses since the pathological process of neurodegeneration is complex. Preferable are natural products and their bioactive components that display neuroprotective

effects through a variety of mechanisms of action. Furthermore, the neuroprotective effects of natural products and their bioactive constituents are significantly influenced by their capacity to pass the blood-brain barrier. Therefore, to increase the neuroprotective action of natural products and their bioactive compounds for the prevention and treatment of neurodegenerative diseases, new approaches and strategies are required, such as the use of nanotechnology in the delivery of natural products and compounds that aid in promoting the access of neuroprotective to the brain.

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