Interdisciplinary Journal of the African Alliance For Research, Advocacy & Innovation
ISSN (O) : 3093-4664
Vol.2. Issue 2 | April-June 2026 www.ijaarai.com

Plant-Derived Bioactive Compounds as Potential Therapeutics in Neurodegenerative Diseases: A Pharmacological Insight

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Abstract

Plant-derived bioactive compounds have provided an attractive and versatile approach to address the challenge of neurodegenerative diseases that remain a critical global health concern due to progressive progression and lack of therapeutic treatment. These diseases include Alzheimer, Parkinson, and Huntington and are characterized by complex pathologies and include oxidative stress, abnormal protein aggregation, mitochondrial dysfunction, neuroinflammation, and neuronal apoptosis that eventually lead to impaired cognition, mobility, and general neurobiological functioning. The traditional pharmacological treatment methods mainly offer relief in the form of symptoms and have a minimal effect of slowing down the process of disease, which is why new forms of treatment are urgently needed. The recent pharmacological studies have progressively emphasized the strong medicinal potential of plant bioactive components, including polyphenols, alkaloids, flavonoids, terpenoids, and polysaccharides. These constituents show a biomolecular range of neuroprotective effects such as antioxidant, anti-inflammatory, anti-apoptotic, anti-cholinesterase as well as anti-aggregation which are direct antagonists of the pathological processes of neurodegeneration.

Consistently, both preclinical in vitro and in vivo experiments reveal that these secondary metabolites are capable of alleviating neuronal damage through the effect of modulating cellular

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ISSN (O) : 3093-4664

signalling pathways, reactive oxygen species, supporting mitochondrial activity, and inhibition of apoptotic cascades. Bacosides (derived with Bacopa monnieri), acteoside, and pinocembrin have been found to, respectively, inhibit neuronal apoptosis, confer anti-inflammatory and antioxidant properties critical to neuroprotection. The growing body of evidence that functional synergy exists between plant-derived compounds and current pharmacotherapies indicates that combination strategies could be used to improve therapeutic outcomes and elevate clinical outcomes.

Keywords: Plant-derived compounds, Bioactive phytochemicals, Neurodegenerative diseases, Alzheimer's disease, Parkinson's disease, Huntington's disease.

1.Introduction

Neurodegenerative diseases, such as Alzheimer disease (AD), Parkinson disease (PD), Huntington disease (HD), and amyotrophic lateral sclerosis (ALS) are one of the health problems that pose a significant global health burden because of their progressive, debilitating character, and the absence of effective curative therapies. They are typified by selective neuronal degeneration, protein misfolding, mitochondrial impairment, oxidative stress, neuroinflammation, and impaired neurotransmitters, all of which join up to destabilize brain homeostasis. Existing pharmacological treatment is more symptomatic than disease-modifying and thus requires new treatment options.

Over the past few years, there has been a growing interest in plant-derived bioactive compounds, e.g. polyphenols, alkaloids, terpenoids, flavonoids, and saponins, due to their pleiotropic effects in pharmacology [1]. The natural compounds are effective antioxidants, anti-inflammatory, anti-apoptotic and neuroprotectants, thus have a potential to be useful in treating the multifactorial pathology of neurodegeneration [2]. An example is curcumin (Curcuma longa), which exhibits sirtuin signalling and mitochondrial biogenesis by binding to amyloid and being an anti-inflammatory molecule. Likewise, clinical adaptation of phytochemicals has been strengthened by the conversion of alkaloids such as galantamine into AD [3]. New pharmacological findings indicate that most plant bioactives are not only suppressing the oxidative stress and neuroinflammation, but also controlling the autophagy, synaptic plasticity, and neuron survival mechanisms, implying that they are useful as disease-modulating agents [4]. Furthermore, research in drug delivery systems and nanotechnology is transcending issues of bioavailability and bio membranes permeability, which continues to reinforce the translational applicability of phytoconstituents in neurotherapeutics [5]. The purpose of this review is to critically assess the importance of plant derived bioactive compounds in terms of neurodegenerative diseases, their pharmacological effects, therapeutic potential and current difficulties in clinical translation [6]. Through a combination of preclinical and clinical evidence, we emphasize the promise of phytochemicals as multi targeted agents in the prevention and treatment of neurodegeneration [7].

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2.Alzheimer's Disease (AD)

2.1 Pathophysiology

Alzheimer Disease (AD) is a progressive neurodegenerative disease, which is associated with several pathological processes that cumulatively lead to mental impairment and neuronal loss. Amyloid-b (Ab) peptides are the hallmark characteristics of AD due to their formation as extracellular aggregates known as b-amyloid plaques. These plaques impair normal communication between neurons, disrupt synaptic activity and cause chronic inflammation by activating microglia. The other important pathological characteristic is the appearance of neurofibrillary tangles (NFTs) in the neurons due to abnormal hyperphosphorylation of tau protein. Tau in its usual state stabilizes, or anchors microtubules that play a crucial role in the transportation of axons, whereas when hyperphosphorylated, the tau proteins dissociate, misfold, and aggregate, causing disrupted transportation within the cell, and eventual cell death [8]. Besides, oxidative stress is also an important AD pathogen. When the generation of reactive oxygen species (ROS) and the antioxidant responses of the brain are imbalanced, oxidative injuries to the neuronal lipids, proteins, and DNA occur [9]. The resulting oxidative damage increases mitochondrial dysfunction and further causes neurodegeneration. Collectively, these pathological mechanisms, amyloid plaque deposition, tau-related neurofibrillary tangles, and oxidative stress, form a toxic environment in the brain, which increasingly damages memory, reasoning, and general cognitive functioning in people with Alzheimer Disease [10].

2.2. Current Treatment Options

The existing medications used in the treatment of Alzheimer disease and related dementias are aimed at symptomatic relief as well as at disease modification. Inhibitors of cholinesterase donepezil, rivastigmine, and galantamine work by raising the level of acetylcholine in the brain and therefore improve cognition, memory and daily functioning. The other pharmacological solution is memantine, an NMDA receptor antagonist, that functions to control glutamatergic activity, and, consequently, to reduce excitotoxicity and improve cognitive and behavioural symptoms [11]. More recent disease-modifying therapies have now been developed including anti-amyloid monoclonal antibodies like aducanumab which bind beta-amyloid plaques in the brain and also aim to slow the progression of the disease. Cognitive stimulation and training exercises, as well as supportive care, are essential components of pharmacological methods and are used to maintain function, behavioural and psychological symptoms, and full-time caregiver support to decrease the emotional and physical burden to the family. All of these strategies are designed to enhance patient quality of life, in addition to reducing the deterioration of functional capabilities and assisting caregivers during the duration of the disease [12].

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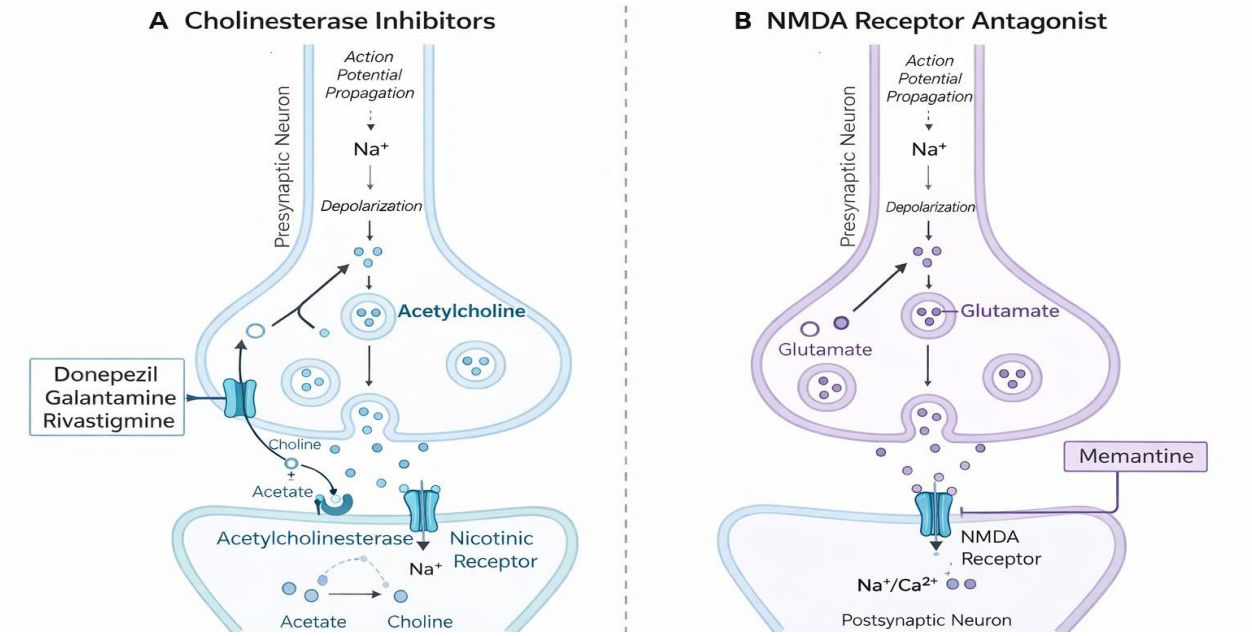

Fig. 1: Mechanism of Action of Alzheimer's Disease Drugs: Cholinesterase inhibitors and NMDA Receptor Antagonist

Figure 1. Mechanism of action of currently approved Alzheimer's disease drugs. (A) Cholinesterase inhibitors (donepezil, rivastigmine, galantamine) enhance cholinergic transmission by inhibiting acetylcholinesterase. (B) Memantine, an NMDA receptor antagonist, reduces glutamate-mediated excitotoxicity by limiting excessive calcium influx.

3.Parkinson's Disease (PD)

3.1 Pathophysiology

Parkinson disease is a progressive neurodegenerative disease mainly due to the loss of dopaminergic neurons in the substantia nigra resulting in low levels of dopamine and poor control of motor control systems in the basal ganglia [13]. One of the critical pathological characteristics is the presence of Lewy bodies (primarily aggregates of misfolded a-synuclein that interfere with cellular functions and leads to the death of neurons). Mitochondrial dysfunction additionally decreases energy generation and augments oxidative stress, which harms neuronal components and accelerates degeneration with neuroinflammation enhancing this harm [14]. A combination of these processes forms the basis of the typical motor (tremor, rigidity, bradykinesia, postural instability), and non-motor (such as decreased speech) symptoms of Parkinsonism [15].

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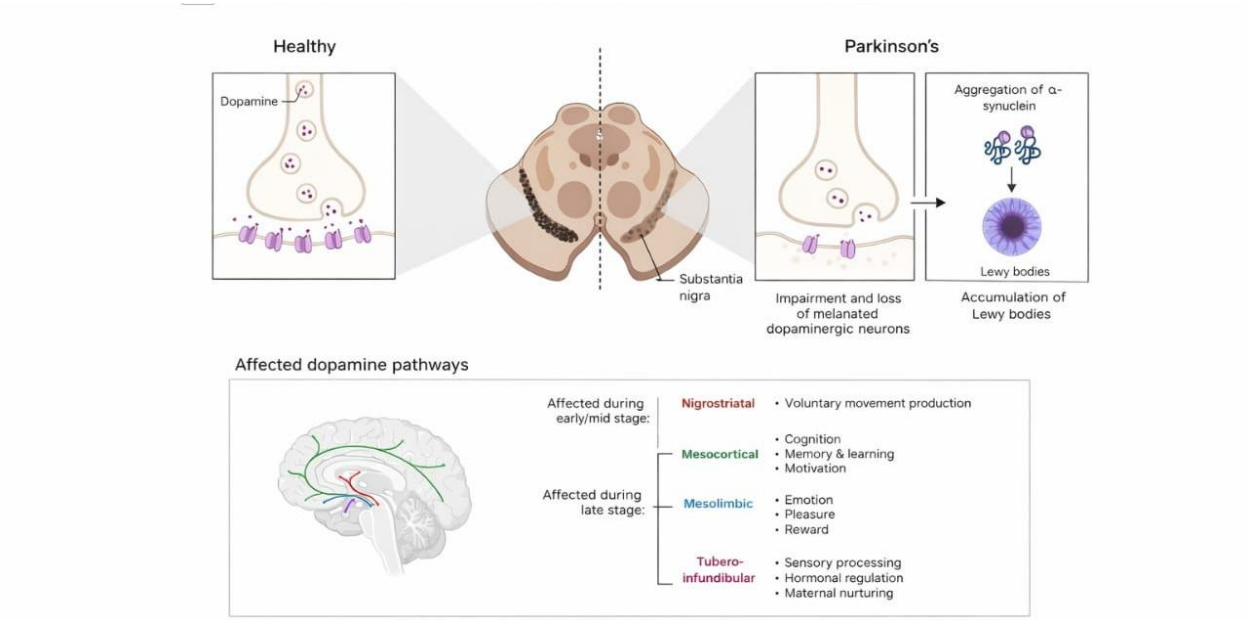

Figure:2 Progression of Parkison's Disease in the Substantia Nigra

Figure 2. Pathophysiology of Parkinson's disease showing degeneration of dopaminergic neurons in the substantia nigra, dopamine depletion, Lewy body formation, and associated motor and non-motor symptoms.

3.2 Current Principle Treatments

Dopamine receptors are directly activated by dopamine agonists like pramipexole and ropinirole and by MAO-B inhibitors like selegiline and rasagiline which slow down the degradation of dopamine extending its effect [16]. In rare and severe cases or where medication is no longer effective, a procedure known as deep brain stimulation (DBS) may be used to enhance the ability to control movement. Physiotherapy is also part of supportive management to ensure that mobility and functionality is maintained and that non-motor symptoms like depression, cognitive impairment, and sleep disturbance are specifically addressed to enhance the overall quality of life [17].

4. Huntington's Disease (HD)

4.1 Pathophysiology

Huntington disease is a hereditary neurodegenerative condition resulting from mutation in the HTT gene, in which an expanded CAG trinucleotide repeat generates the mutant huntingtin protein (mHTT) having toxic gain-of-function properties [18]. The characteristic motor, cognitive and psychiatric symptoms are the resultant of progressive loss of medium spiny GABAergic neurons in the striatum,

ISSN (O) : 3093-4664

particularly in the caudate and putamen [19]. The mutant huntingtin is aggregated intranuclearly and cytoplasmically, affecting transcriptional control, mitochondrial energy metabolism, excitotoxicity via glutamate, the ubiquitin-proteasome degradation pathway, and neuroinflammation. As the disease advances, neurodegeneration is seen not only to the striatum but also to the cortex, globus pallidus, cerebellum, amygdala, and hippocampus leading to a generalized neurological dysfunction [20].

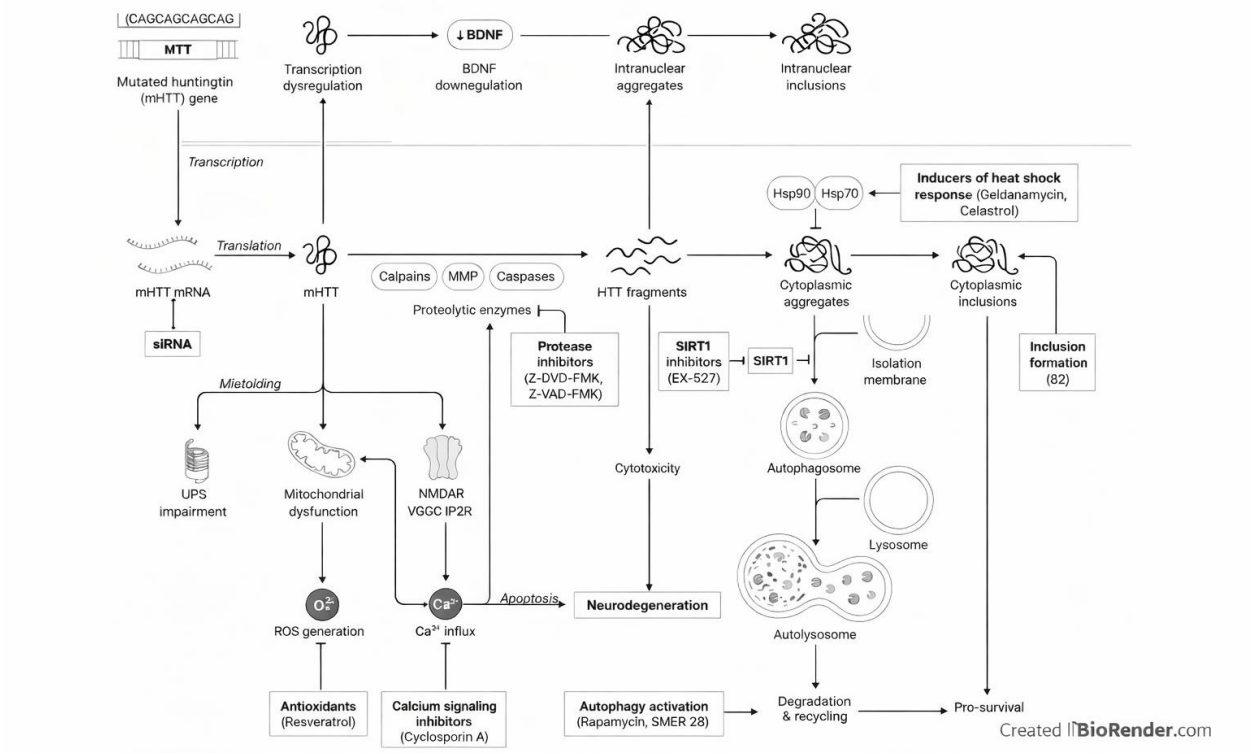

Fig 3. Huntington's Disease Pathogenesis

Figure 3. Molecular pathogenesis of Huntington's disease illustrating mutant huntingtin aggregation, transcriptional dysregulation, mitochondrial dysfunction, excitotoxicity, impaired proteostasis, and neuronal degeneration.

4.2 Current treatment options

Huntington disease cannot be cured nowadays and can only be treated symptomatically and supportively. The primary pharmacologic choices to treat motor symptoms, i.e., chorea, are tetrabenazine and

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deutetrabenazine, and psychiatric symptoms are treated with mood stabilizers, antidepressants (SSRI/SNRI), and antipsychotics [21]. Non-pharmacologic interventions that are used to preserve functioning and increase quality of life include: psychotherapy, physical therapy, occupational therapy and speech therapy [22]. New disease-modifying strategies are in development, including antisense oligonucleotides (ASOs) and RNA interference (RNAi) to silence the production of mutant huntingtin, as well as pre-clinical or experimental technologies, such as stem cell therapy, gene editing technologies (CRISPR, TALEN, ZFP), and neuroinflammatory drugs, but all of these are in development [23].

5. Classification of Plant-Derived Bioactive Compounds

5.1 Polyphenolic Compounds

The most well-known and extensively studied group of in-plant bioactive constituents are the polyphenols, which are characterized by several phenolic hydroxyl groups joining their aromatic rings [24]. The class is further divided into a number of subclasses with distinct chemical skeletons and bioactivities:

5.1.1 Flavonoids: Flavonoids are also one of the most common polyphenols, which are quercetin, kaempferol, and epigallocatechin gallate, all of which have proven strong antioxidant, anti-inflammatory, and metal-chelating activity. They have a high potential of targeting neurological and cardiovascular diseases, especially since they are able to modulate cell signalling pathways and interact with metal ions [25].

5.1.2 Phenolic Acids: These are compounds, e.g., caffeic and ferulic acid that are known to possess powerful radical-scavenging properties, and have been reported to regulate inflammation and prevent oxidative injury [26].

5.1.3 Stilbenes: Resveratrol is the most prominent member and is present in grapes and berries, and it has been receiving attention due to its versatile neuroprotective actions including sirtuin protein activation (especially SIRT1), regulation of inflammatory pathways, and suppression of oxidative stress.

5.1.4 Lignans: Lignans are antioxidant and estrogenic compounds that are present in seeds and whole grains, add to their effects on hormone regulation and disease prevention [27].

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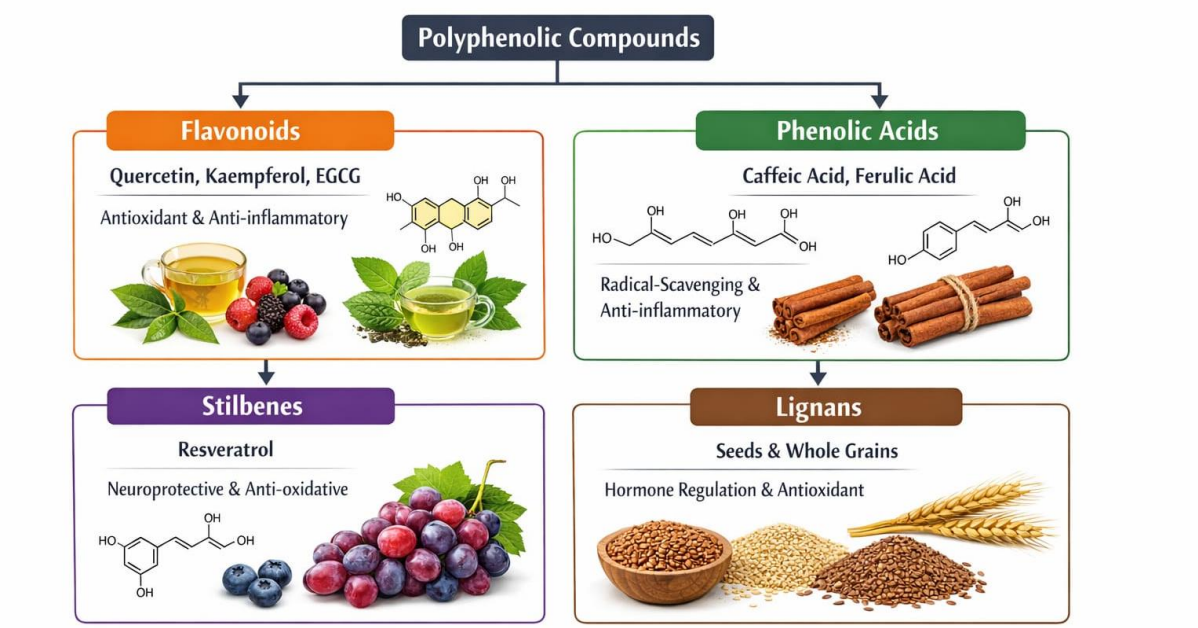

Fig 4. Classification of Plant -Derived Bioactive Compounds.

5.2 Curcumin

The main polyphenol of turmeric (Curcuma longa) has become a paradigmatic exemplar of bioactive phytochemicals with wide-spectrum pharmacological applications, i.e. curcumin [28]. Its neuroprotective effects include the prevention of nuclear factor-kB (NF-kB) inflammatory pathway, the suppression of the production of pro-inflammatory cytokines, as well as, its mechanism of preventing aggregation of amyloid-b peptides which is a characteristic of neurodegenerative pathologies like the Alzheimer disease. Although curcumin has a promising future in terms of therapy due to the comprehensive preclinical research on this compound, its low bioavailability, and the lack of potential to pass the blood-brain barrier, complicate its clinical efficacy, prompting the creation of sophisticated delivery models [29].

5.3 Resveratrol

The stilbene Resveratrol is an interesting compound with a very impressive neuroprotective ability due to the ability to transcribe sirtuin enzymes (SIRT1 in particular) [30]. This measure allows this to influence metabolic control, cellular lifespan, and neuroinflammation]. This measure allows this to influence metabolic control, cellular lifespan, and neuroinflammation. Yet, clinical studies are inconclusive and some studies report cognitive and other beneficial effects on disease biomarkers, whereas other studies do not report any significant therapeutic benefit [31].

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5.4 Quercetin

Quercetin is very common in plant life and an effective antioxidant and a moderate blood-brain barrier penetrant. Its pharmacological property is also supplemented by the capacity to chelate transition metals as well as mitigate oxidative damage which places it as the desirable candidate in neurological and vascular protection [32].

Table 1. Polyphenolic compounds and their neuroprotective pharmacological properties.

Subclass / Compound		Plant Source / Example	Key Pharmacological Actions	Relevance in Neurodegenerative Diseases	Reference
Flavonoids (Quercetin, Kaempferol, EGCG)		Onions, apples, tea, green tea	Antioxidant, anti-inflammatory, metal chelation, signaling modulation	AD, PD, vascular dementia	33
Phenolic acids (Caffeic acid, Ferulic acid)		Coffee, fruits, whole grains	Radical scavenging, anti-inflammatory, oxidative injury prevention	Neuroinflammation, oxidative stress-related ND	34
Stilbenes (Resveratrol)		Grapes, berries, red wine	SIRT1 activation, anti-inflammatory, antioxidant, mitochondrial protection	AD, PD, age-related decline	35
Lignans		Flaxseed, sesame, whole grains	Antioxidant, estrogenic activity, hormone regulation	Hormone-related neuroprotection	36
Curcumin		Turmeric (Curcuma longa)	Anti-amyloidogenic, NF-κB inhibition, cytokine suppression, antioxidant	AD, PD (low bioavailability is a limitation)	37

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Quercetin		Apples, onions, tea	BBB penetrant, antioxidant, metal chelation	AD, PD, vascular neuroprotection	38

5.5 Alkaloids

Another central type of plant bioactive agent is alkaloids, which are defined by the presence of one or more nitrogen atoms in the chemical structure. Alkaloids are nitrogen-containing compounds with a variety of biological activities, such as neuroprotection, antioxidant, anti-inflammatory and enzyme inhibition [39]. These substances have strong biological actions in the central nervous system and in peripheral tissues:

5.5.1 Galantamine and Huperzine A: both are well-known plant-based acetylcholinesterase inhibitors, and are successfully approved by regulators to be used in the treatment of Alzheimer disease because of their effectiveness in enhancing cholinergic neurotransmission and cognitive capabilities [40].

5.5.2 Berberine: Derived in the genus Berberis, berberine has anti-inflammatory and neuroprotective properties most likely due to its inhibition of the NLRP3 inflammasome pathway, and as a result, has therapeutic potential in neuroinflammatory diseases [41]. Berberine is able to enhance memory, cognitive, and motor function in animal disease models via regulation of neurotransmitter levels and synaptic health [42].

5.6 Terpenoids

The terpenoids or isoprene-based compounds are an enormous family of compounds that are made up of units of isoprene. Many terpenoids have antioxidant, anti-inflammatory, and anti-apoptotic effects to protect neuronal cells from damage and facilitate recovery after injury or neurodegeneration [43]. Their structural variety leads to a large variety of physiological effects:

5.6.1 Ginkgolides: Ginkgolides are putative cognitive-enhancing extracts derived out of Ginkgo biloba whose clinical outcomes are inconclusive, and people argue whether they can prevent cognitive decline and neuroprotection [44].

5.6.2 Ginsenosides: Ginsenosides are derived products of Panax ginseng, and have some neuroprotective, antioxidant and growth factor-modulating activity, which explains their wide usage in traditional medicine and as dietary supplements to support the health of the brain [45]. Ginsenosides are the main bioactive compounds of ginseng and have important antioxidant, anti-inflammatory, immuno-modulatory and anti-cancer properties.

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Table 2. Alkaloids and Terpenoids with neuroprotective potential.

Compound Class	Representative Compounds	Source Plants	Mechanism of Action	Neurodegenerative Application	Reference
Alkaloids	Galantamine, Huperzine A	Galanthus (snowdrop), Huperzia serrata	Acetylcholinesterase inhibition, enhances cholinergic neurotransmission	Alzheimer's disease (FDA/CFDA approved)	[46]
	Berberine	Berberis spp.	NLRP3 inflammasome inhibition, anti-inflammatory, antioxidant	Neuroinflammation, AD, PD	46
Terpenoids	Ginkgolides, Bilobalide	Ginkgo biloba	Antioxidant, anti-apoptotic, improves cerebral blood flow	AD, vascular dementia	[47]
	Cannabinoids (CBD, THC)	Cannabis sativa	Endocannabinoid receptor modulation, neuroprotection, anti-excitotoxic	PD, MS, neuroinflammation	[47]
	Asiaticoside	Centella asiatica	Enhances neuronal plasticity, antioxidant	Cognitive impairment, AD	[47]

5.7 Carotenoids and Fatty Acids

Carotenoids like astaxanthin are of great importance due to their unrivaled antioxidant capacity and free radical-scavenging properties. Astaxanthin is a water-soluble antioxidant, present in seafood and algae, which has been proven to be superior to most other antioxidants in its capacity to prevent oxidative damage on cell structures [48]. Fatty Acids especially omega-3 polyunsaturated fatty acid, such as, Docosahexaenoic acid (DHA) have essential functions in the neuronal membrane fluidity, in the regulation of inflammatory events, and in neuroprotection.

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These bioactive lipids play an essential role in the development of the brain, synaptic plasticity and in preventing neurodegenerative diseases [49].

6. Molecular Mechanism of Neuroprotection

6.1 Oxidative Stress Mitigation

The susceptibility of the brain to oxidative stress is due to the fact that the brain has a disproportionate high oxygen consumption, high polyunsaturated fatty acids, and has a relatively weak inherent antioxidant defence [50]. Aging causes oxidative damage that is the basis of numerous neurodegenerative diseases. The oxidative stress can be alleviated by plant-derived compounds, including flavonoids, polyphenols, and isothiocyanates, via various mechanisms that may be classified as direct neutralization of free radicals (scavenging), redox-active metals chelation (preventing the formation of radicals), and, most notably, the stimulation of endogenous antioxidant signalling [51]. The signalling pathway of central importance in this effect is the Nrf2 signalling pathway. When electrophilic phytochemicals such as sulforaphane activate it, Nrf2 is translocated to the nucleus, where it binds to antioxidant response elements (AREs) in DNA [52]. This increases the expression of essential enzymes including superoxide dismutase (SOD), catalase, and glutathione peroxidase which strengthens the ability of the cell to detoxify reactive oxygen species (ROS). An example of a well-documented Nrf2 activator is its sulforaphane, which is present in broccoli and other crucifers, although numerous flavonoids and phenolic acids demonstrate a comparable but weaker effect [53].

6.2 Neuroinflammation Modulation

Chronic neuroinflammation characterized by the activation of microglia and maintained by cytokines such as TNF-a, IL-1b, and IL-6 is the pathogenesis of many neurological diseases [54]. Phytochemicals are capable of reducing this inflammatory condition through their effect on the NF-kB pathway and other associated transcription factors stimulating the expression of pro-inflammatory genes. Due to its extensive anti-inflammatory effect, including the inhibition of cytokines but also enzymes such as cyclooxygenase and inducible nitric oxide synthase, curcumin is especially interesting [55]. Resveratrol also demonstrates the multifaceted effect of plant compounds, being capable of altering microglial states - switching M1 (pro-inflammatory) to M2 (anti-inflammatory, repair-promoting) [56]. This phenotypic plasticity is fundamental in the solution of inflammation and repairment of neural homeostasis. In the long-term these interventions can mitigate the rate of neuronal loss and maintenance of cognitive functions [57].

6.3 Protein Aggregation Inhibition

Protein aggregation and misfolding (e.g., amyloid-betain Alzheimer, a-synuclein in Parkinson) are pathological events of neurodegeneration, which contribute to cellular toxicity. Some

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phytochemicals have the ability to stabilize the aberrant proteins in less toxic conformations or even disaggregate already existing toxic aggregates [58]. Green tea polyphenol, epigallocatechin gallate (EGCG), has been shown to divert aggregation of proteins into non-toxic off-pathways, as well as to dissolve established aggregates. Curcumin, also, disrupts the assembly and stability of amyloid fibrils, underpinning prevention and treatment measures [59].

6.4 Mitochondrial Function Enhancement

Mitochondrial dysfunction is a cause and consequence of neurodegeneration: dysfunctional mitochondrion will result in reduced production of ATP and increased ROS, which in turn contribute to the cycle of destruction [60]. Phytochemicals like quercetin and berberine have the possibility to increase the mitochondrial biogenesis and improve functioning by activating the AMP-activated protein kinase (AMPK) and peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1a) [61]. These pathways result in the multiplication of mitochondrial numbers and efficiency. A widely used flavonoid, quercetin, has been of specific interest in animal models in the restoration of mitochondrial capacity, which might cancel age- and disease-associated losses. Berberine, a traditional herbal medicine, also promotes the health of mitochondria, and has a potential indirect effect of overriding metabolic syndromes, which frequently co-morbidly occur with neurodegenerative risk [62].

6.5 Blood-Brain Barrier Permeability and Bioavailability

Many phytochemicals are blocked by the protective blood-brain barrier (BBB) and are therefore not directly efficacious. BBB passage factors consist of molecular size, charge, and lipid solubility, active efflux and influx transporters [63]. A number of approaches are being explored to circumvent these limitations, including lighting it up by methylating to make it lipophilic, and nano formulation (e.g. coating curcumin in lipid nanoparticles) in order to get it to the brain. Another promising route is the transporter-mediated systems such as the conjugation of glucose and amino acids. Furthermore, gut microbiota are capable of converting absorbed phytochemicals into more readily absorbable derivatives and BBB permeable, which demonstrates the interconnectedness of the gut-brain axis and neuroprotection [64].

7. Clinical Evidence and Therapeutic Applications

7.1 Alzheimer's Disease

In humans, compounds, such as resveratrol and curcumin, have gone to clinical trial stages. The safety of resveratrol has been determined to a dose of 2g/day, although with mild but encouraging disease biomarker and cognitive outcome effects [65]. The drawback of curcumin was that it was never absorbed well, but newer preparations are not only demonstrating better bioavailability, but

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also cognitive effects. Ginkgo biloba is still popular and provided modest cognitive benefits in meta-analyses especially in the cases of early or mild cases of Alzheimer [66].

7.2 Parkinson's Disease

Considering the key functions of oxidative and inflammatory stress in Parkinson, plant-derived compounds are reasonable as therapeutic options. Although there is a relative lack of robust clinical data, preclinical evidence of neuroprotection is uniformly reported with polyphenol-rich extracts (e.g., green tea, curcumin, resveratrol) [67]. There is hints in population-level data that polyphenol-rich diets might alleviate the risk of PD, though that this effect is urgently awaited in the rigorous trials [68].

7.3 Combination Therapies

The emerging body of work suggests that the combination of phytochemicals, i.e. curcumin with resveratrol, can be more neuroprotective than the agents singly and enhanced through the effect of absorption and synergism [69]. Those combinations are more efficient in addressing a wide range of pathological mechanisms observed in neurodegeneration (e.g., oxidative damage, inflammation, protein aggregation). It is also indicated with evidence that omega-3 fatty acids together with polyphenols show even stronger effects as anti-inflammatory and cognitive-protective agents, confirming dietary [70].

8. Regulatory and Translation Pathways

Converting positive results in the laboratory to useful clinical practice poses enormous barriers and challenges. Such challenges are especially acute with natural products, which in many cases may actually have several active components that may act through a variety of mechanisms and act synergistically. This complicates the design and execution of clinical studies and complicates the ability to isolate and measure particular effects of therapy [71]. Moreover, the heterogeneity in the structure and biological functionality of natural products necessitates careful standardization and stringent quality measures to be undertaken to assure reproducibility and credibility across the clinical development [72]. The choice of a suitable endpoint is one of the greatest challenges in clinical development, particularly in the early phase of the disease progression [73]. The conventional cognitive tests have served as the foundation of assessing neurodegenerative diseases but tend to be insensitive and unspecific to the ability to identify slight changes that point to the modification of the disease at the earlier stages [74]. In a bid to counter this weakness, biomarker endpoints have become more popular. These incorporate both sophisticated neuroimaging technologies like PET, and MRI scans, and fluid biomarkers that can be measured in cerebrospinal fluid or blood. To track the progression and therapeutic response to disease and enable earlier and more accurate identification of clinical advantages in trials of natural products, biomarkers offer a

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more sensitive and objective method [75].

9.Clinical Development Challenges

There are more complex challenges in the clinical development of natural products in neurodegenerative diseases than the complexity of study design[76]. Variability in plant composition, differences in active ingredient concentrations, and interactions between multiple bioactive compounds complicate dosage standardization and safety assessments. Also, the neurodegenerative disorders are multifactorial, and thus require multifactorial treatment and it is hard to pinpoint clinical effects observed to any particular component of a natural product [77]. Such complexity requires innovative trial designs including adaptive, biomarker-driven and precision medicine approaches. Selection of endpoints is a major challenge particularly in the first stages of diseases where the clinical scales in use at the time could fail to identify subtle improvements in cognition or functioning [78]. The use of biomarker-driven endpoints, such as neuroimaging as well as fluid biomarkers, increases sensitivity to identify biological changes due to disease modification. These endpoints offer important surrogate markers that have the potential to substantially reduce trial time and enhance the predictive validity of clinical outcomes at drug development stages [79].

10. Regulatory Framework Evolution

Governmental bodies of the world are becoming aware of therapeutic possibilities of natural products in treating neurodegenerative conditions [80]. US Food and Drug Administration (FDA) has provided botanical drug guidance that gives a clear-cut regulatory direction to standardized, scientifically validated plant-based therapies. This guideline gives the specifications of the safety, efficacy, and consistent production processes that are required to be shown in complex botanicals [81]. In Europe, the European Medicines Agency (EMA) has come up with guidelines that directly deal with the unique issues that are related to herbal medicinal products. These recommendations consider the fact that the chemical composition is often complicated, conventional use, and scientific data required to assess herbal medicines [82]. These liberal regulatory frameworks contribute to the correction of balancing the safety of patients with the propensity to encourage the development of innovation, which allows natural products to be considered as viable therapeutic agents in standard clinical practice [83].

11. Economic and Societal Implications

Neurodegenerative diseases have an enormous worldwide economic cost of more than 800 billion annually. This value includes direct medical spending, such as hospital and long-term care, drugs and outpatient services. Moreover, the indirect costs linked to lost productivity of the disabled and untimely death, intensive care giving duties by families and the healthcare systems play a major part in this societal

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impact [84]. This burden on the financial resources of millions of patients and their families can be significantly relieved through the development and implementation of effective disease-modifying therapies and the resulting improvement in their quality of life [85]. To date, cost-effectiveness studies of phytochemical and natural product-based interventions demonstrate positive economic characteristics, especially during their application as preventive strategies in the at-risk communities. Their ability to be incorporated into large-scale prevention of diseases and early intervention due to their relatively low production costs, and their possible prospects of being widely accessible and easy to administer underpins the incorporation of natural products in the health policies of populations that aim at large-scale health promotion [86].

12. Challenges and Future Directions

The bioactive compounds derived in plants have been shown to have strong preclinical potential in neurodegenerative diseases, yet their translation has been associated with numerous constraints and issues including poor pharmacokinetics, low bioavailability, limited penetration of the blood-brain barrier, variability in plant extract, limited clinical data, and lack of certainty in long-term safety or drug-drug interactions. In addition, animal and cellular models are only partially representative of the complexity of human disease, and their development is complicated by regulatory and quality-control challenges. The future would be in better delivery systems (e.g. nanotechnology, prodrugs), standardization of extraction and characterization techniques, network pharmacology and AI use to discover multi-target approaches and rigorous biomarker-driven clinical trials. Integration of phytochemicals with other treatments, investigation of less well-studied diseases, such as ALS or Huntington disease, mapping interactions between phytochemicals and toxicology, and sustainable sourcing will further support their role as adjunctive or disease-modifying treatments in neurodegenerative medicine.

13. Conclusion

Plant-derived bioactive compounds have a high potential as a therapy in neurodegenerative diseases since they act on several targets that contribute to these multifaceted disorders. These natural compounds have the capacity to address some of the major processes that contribute to brain cell damage and disease progression and are therefore appropriate to the treatment of such complex conditions. Nevertheless, they still have some hurdles to go through before they can be mass-produced, including how well they are absorbed and utilized by the body, the consistency in quality and strength across the batches, and whether they can translate findings on their use in laboratory into the real-world clinical practice. Fortunately, these challenges can be overcome with advances in formulation technologies to enhance delivery, research into personalized medicine approaches, and changing regulatory standards to provide better treatments to people.

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ISSN (O) : 3093-4664

With the active development of research on neurodegeneration, the importance of plant-based bioactive compounds in the treatment regimen would increase. These herbal products perfectly combine the ancient knowledge of traditional medicine and the accuracy and objectivity of contemporary scientific studies. A combination of these methods can alter the way the neurodegenerative diseases are managed and yield positive results, providing a better quality of life to the existing patients, and to the upcoming generation that may struggle with these illnesses. This integrative therapy has great potential in influencing the future management of neurodegenerative diseases in a positive and meaningful manner.

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