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Role of *Tinospora cordifolia* Extracts in Neurodegenerative Disorders

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Abstract: *Tinospora cordifolia* (Willd.), commonly known as Guduchi, has attracted significant attention for its potential neuroprotective properties. Several studies have demonstrated the effectiveness of *Tinospora cordifolia* and its extracts in mitigating neurodegenerative diseases, such as Alzheimer's and Parkinson's, which are characterized by progressive neuronal degeneration leading to cognitive decline and motor dysfunction. The neuroprotective potential of *Tinospora cordifolia* is attributed to its rich repertoire of bioactive compounds, including alkaloids, glycosides, steroids, and polysaccharides, which exhibit potent antioxidant, anti-inflammatory, and anti-apoptotic effects essential for preventing neuronal damage. Experimental evidence indicates that *Tinospora cordifolia* can enhance cognitive function, reduce oxidative stress, and suppress neuroinflammation. Moreover, it modulates critical signaling pathways involved in neuronal survival and synaptic plasticity, further supporting its therapeutic potential. Additional mechanisms include metal ion chelation, inhibition of amyloid-beta accumulation, and enhancement of cholinergic function, all of which are relevant to neurodegenerative pathology. Despite promising preclinical findings, clinical evidence remains limited, highlighting the need for rigorous clinical trials to confirm both efficacy and safety in humans. These investigations are essential to establish *Tinospora cordifolia* as a viable natural therapy for neurodegenerative diseases.

Keywords: *Tinospora cordifolia*, Antioxidant, Neurodegeneration, Neuroinflammation, Neuroprotective.

1. Introduction

Neurodegenerative diseases are a broad category of disorders characterized by the progressive structural or functional loss of cells in the nervous system. These diseases primarily affect neurons, the fundamental building blocks of the brain and spinal cord. Neurodegenerative disorders are generally classified into well-established conditions such as Alzheimer’s disease (AD), Parkinson’s disease (PD), Huntington’s disease (HD), and amyotrophic lateral sclerosis (ALS), while relatively newer identified conditions include frontotemporal dementia (FTD) and multiple system atrophy (MSA) [1].

1.1 Alzheimer's Disease (AD)

Alzheimer’s disease (AD) is the most common form of dementia, accounting for approximately 60–80% of all dementia cases. It is characterized by the accumulation of amyloid plaques and neurofibrillary tangles, which lead to the death of neurons and the loss of synaptic connections. The disease typically begins with memory loss and confusion and gradually progresses to severe cognitive decline, loss of bodily function control, and ultimately death. While the exact cause of AD remains unclear, it is believed to result from a combination of genetic, environmental, and lifestyle factors. Current therapeutic strategies primarily involve cholinesterase

inhibitors and NMDA receptor antagonists, which can alleviate symptoms but do not halt disease progression [2].

1.2 Parkinson's Disease (PD)

Parkinson's disease (PD) is a neurological disorder primarily affecting the motor system, caused by the loss of dopamine-producing neurons in the substantia nigra region of the brain. This neuronal depletion leads to the characteristic motor symptoms of PD, including tremors, rigidity, bradykinesia, and postural instability. In addition to motor impairments, patients may experience non-motor symptoms such as dementia, depression, and other mood disorders. The mainstay of treatment involves dopamine replacement therapy, most commonly with levodopa, and in advanced cases, deep-brain stimulation. While these interventions can stabilize symptoms and improve quality of life, they do not halt the progression of the disease [3].

1.3 Huntington's Disease (HD)

Huntington's disease (HD) is an inherited neurodegenerative disorder caused by a genetic mutation on chromosome 4 in the HTT gene, which encodes the huntingtin protein. Mutant huntingtin primarily causes the death of neurons in the basal ganglia, a brain region responsible for regulating movement, cognition, and emotions. Clinically, HD is characterized by motor dysfunction (chorea), cognitive decline, and psychiatric disturbances. The disease typically manifests in mid-adulthood and progresses over 15–20 years. Currently, there is no cure for HD, and treatment is largely symptomatic: chorea may be managed with tetrabenazine, while psychiatric symptoms are treated with antipsychotic medications [4].

1.4 Amyotrophic Lateral Sclerosis (ALS)

Amyotrophic lateral sclerosis (ALS), commonly known as Lou Gehrig's disease, is a neurodegenerative disorder characterized by the progressive loss of motor neurons in the brain and spinal cord. This neuronal damage leads to muscle weakness, atrophy, and paralysis, ultimately resulting in loss of speech, difficulty swallowing, and respiratory failure. Approximately 20% of ALS cases are linked to a family history, while the remaining ~90% are sporadic (idiopathic). Currently, riluzole and edaravone are the only FDA-approved medications which offer modest benefits in slowing disease progression. Supportive and complementary interventions, such as physiotherapy and respiratory care, are essential for symptom management and improving quality of life, although they do not alter the overall prognosis [5].

1.5 Fronto-temporal Dementia (FTD)

Frontotemporal dementia (FTD), also known as Pick's disease, comprises a group of disorders caused by the degeneration of neurons in the frontal and temporal lobes of the brain. Clinically, FTD is characterized by changes in personality, behavior, and language, reflecting the regions of the brain affected. The disease typically has a gradual onset and usually manifests between the ages of 40 and 65 years. Symptoms vary widely among individuals and are often atypical, making diagnosis challenging. Currently, there is no cure for FTD, and treatment primarily focuses on managing symptoms through pharmacological approaches and psychosocial interventions [6].

1.6 Multiple System Atrophy (MSA)

Multiple system atrophy (MSA) is a rare neurodegenerative disorder that affects motor control, autonomic functions, and other bodily systems. The disease is classified into two subtypes based on predominant symptoms: MSA-P, characterized mainly by Parkinsonian features, and MSA-C, marked primarily by cerebellar manifestations. MSA is associated with rapidly progressive disability, with most patients experiencing severe functional impairment within a few years of disease onset. Currently, there is no cure for MSA, and treatment is focused on symptomatic relief and improving quality of life [7].

2. Pathophysiology and Etiology

The pathophysiology of neurodegenerative diseases is complex and multifactorial, making it challenging to fully elucidate. Common pathological processes include protein misfolding and aggregation, mitochondrial dysfunction, oxidative stress, excitotoxicity, and neuroinflammation. In specific diseases, these processes are

associated with characteristic proteins: amyloid-beta and tau in Alzheimer's disease (AD), alpha-synuclein forming Lewy bodies in Parkinson's disease (PD), mutant huntingtin in Huntington's disease (HD), and various mechanisms, including RNA metabolism dysfunction and protein aggregation in amyotrophic lateral sclerosis (ALS). Genetic factors play a significant role in certain neurodegenerative disorders. Known gene mutations include APP, PSEN1, and PSEN2 in AD; LRRK2 and PARK2 in PD; HTT in HD; and SOD1 and C9orf72 in ALS. However, the majority of neurodegenerative diseases are sporadic, arising from a combination of environmental factors, toxins, infections, lifestyle choices, and aging [8]. These multifactorial influences contribute to neuronal vulnerability and the progressive nature of these disorders.

3. Diagnosis and Biomarkers

The diagnosis of neurodegenerative disorders requires a combination of clinical assessment, imaging, and biomarker evaluation. In Alzheimer's disease (AD), biomarkers include measurements of amyloid-beta and tau in the cerebrospinal fluid, alongside PET imaging. Parkinson's disease (PD) is primarily diagnosed clinically, often supported by dopamine transporter imaging. In Huntington's disease (HD) and familial ALS (FALS), genetic testing aids molecular diagnosis and validates clinical findings. Current research continues to focus on identifying accurate biomarkers for early detection, disease staging, and monitoring therapeutic response [9].

4. Future Directions

Current research emphasizes understanding disease etiology, the development of disease-modifying therapies, and improved diagnostics. Advances in genetics, molecular biology, and neuroimaging hold promise for early detection, personalized medicine, and targeted interventions. Efforts include the development of disease-modifying drugs targeting amyloid-beta and tau in AD, alpha-synuclein in PD, and mutant huntingtin in HD. Concurrently, neuroprotective strategies, stem cell therapies, and gene-editing approaches such as CRISPR-Cas9 are being explored as potential therapeutic targets [10].

5. The significance of Herbal remedies, specifically in neurodegenerative disorders

Neurodegenerative diseases are a rapidly growing contributor to the global burden of disease, affecting the structural and functional integrity of neurons. Conditions such as AD, PD, and HD not only severely impair the quality of life of patients but also pose significant challenges to healthcare systems worldwide. Current conventional therapies primarily alleviate symptoms without altering disease progression, which has led to increased interest in alternative and complementary treatments, including herbal remedies. Herbs and plant-derived compounds are being investigated for their potential to modulate disease pathways, provide neuroprotection, and support neuronal function, offering a promising avenue for disease-altering interventions [11].

5.1 Historical Perspective

Herbal medicine has a rich and longstanding history, with records of usage spanning multiple civilizations and generations. Many medicinal plants contain diverse phytochemicals, including antioxidants, anti-inflammatory agents, and other bioactive compounds. Recently, there has been growing scientific interest in herbal therapies as potential neuroprotective agents, driven by the need for novel treatments for neurodegenerative diseases [12].

5.2 Mechanisms of Action

Herbal remedies are believed to exert neuroprotective effects by modulating multiple molecular pathways involved in neurodegeneration. For instance, curcumin, a bioactive compound derived from turmeric (*Curcuma longa*), exhibits potent anti-inflammatory and antioxidant properties, helping to mitigate oxidative stress and neuroinflammation, which are central contributors to neuronal damage and disease progression [13].

5.3 Evidence from Research

Recent studies provide substantial evidence that several herbal medicines may be effective in neurodegenerative conditions (**Table 1**). Some key examples include:

5.3.1 *Ginkgo biloba*

Extracts from *Ginkgo biloba* leaves have demonstrated neuroprotective effects through their antioxidant activity and ability to inhibit amyloid-β formation, a key factor in Alzheimer's disease pathology [14].

5.3.2 *Bacopa monnieri*

Known for its cognitive-enhancing properties, *Bacopa monnieri* has been studied for its potential to improve memory and cognitive function in models of neurological disorders [15].

5.3.3 Ashwagandha (*Withania somnifera*)

An adaptogenic herb, Ashwagandha, has been shown to reduce neuroinflammation and enhance neuronal plasticity in animal models of neurodegenerative diseases, suggesting its potential as a therapeutic agent [16].

Table 1: Herbal Plants and Their Neuroprotective Effects.

Herbal Plant	Mechanism of Action	References
Ginkgo biloba	Antioxidant, anti-inflammatory, neuroprotective	[17]
Curcumin (*Curcuma longa*)	Anti-inflammatory, neuroprotective	[18]
Bacopa monnieri	Enhances cognitive function, antioxidant	[19]
Withania somnifera (Ashwagandha)	Reduces stress, enhances neuronal regeneration	[20]
Gotu kola	Improves memory, enhances cerebral circulation	[21]
Huperzine A	An acetylcholinesterase inhibitor enhances neurotransmission	[22]

6. Herbs and Their Antidote in Neurodegenerative Disorders

Herbs have been valued for centuries in traditional medical systems, including Ayurveda in India, Traditional Chinese Medicine (TCM), and various Western traditional practices, such as Native American medicine. The therapeutic potential of these herbs is only now being increasingly recognized by modern science, particularly in the context of neurodegenerative diseases—including Alzheimer's disease, Parkinson's disease, Huntington's disease, and amyotrophic lateral sclerosis (ALS). These conditions are characterized by the progressive deterioration of the structure and function of the nervous system, leading to cognitive decline, motor dysfunction, and other pathological manifestations [20]. The following sections highlight the significance of herbal remedies in the management and neuroprotection of these disorders:

6.1 Antioxidant Properties

Many herbs possess strong antioxidant activity, which helps counteract free radicals, a major contributor to oxidative stress in neurons. For example, flavonoids and terpenoids present in *Ginkgo biloba* demonstrate significant antioxidant potential, protecting neurons from oxidative damage [23].

6.2 Anti-inflammatory Effects

Chronic neuroinflammation is a common feature of neurodegenerative diseases. Certain herbs, such as *Curcuma longa*, contain curcumin, which exhibits potent anti-inflammatory effects. Curcumin has been shown to inhibit microglial and astrocyte activation, thereby mitigating inflammation-associated neuronal damage [24].

6.3 Neuroprotective Compounds

Some herbs contain bioactive molecules that provide direct neuroprotection, preventing neuronal degeneration. *Bacopa monnieri* (Brahmi), for instance, has demonstrated anti-neurodegenerative properties, enhancing neuronal health and improving learning and memory [25].

6.4 Modulation of Neurotransmitters

Certain herbal compounds can influence neurotransmitter levels and activity, which is beneficial in managing neurodegenerative symptoms. For example, St. John's Wort (*Hypericum perforatum*) modulates serotonin levels, making it useful in alleviating depressive symptoms associated with neurodegenerative disorders [26].

7. Benefits of Specific Herbs

7.1 *Ginkgo Biloba*

Ginkgo biloba is a herbal extract known to enhance cognition and increase cerebral blood flow, while also exhibiting antioxidant and anti-inflammatory properties. It has been studied for its potential to improve cognitive impairment in Alzheimer's disease patients [27].

7.2 Turmeric (*Curcuma longa*)

Curcumin, the active compound in *Curcuma longa*, possesses potent anti-inflammatory and antioxidant effects. In addition to vascular benefits in conditions like erectile dysfunction, curcumin has been shown to reduce amyloid plaque formation in Alzheimer's disease, highlighting its neuroprotective potential [28].

7.3 *Bacopa monnieri* (Brahmi)

Bacopa monnieri is widely used to enhance memory, particularly in individuals with cognitive challenges. Its beneficial effects may result from improved synaptic transmission and antioxidant-mediated neuroprotection [29].

7.4 Ashwagandha (*Withania somnifera*)

Ashwagandha has adaptogenic properties that help manage stress and anxiety, factors known to exacerbate neurodegenerative diseases. It also supports neuronal regeneration and reduces oxidative stress, contributing to neuroprotection [30].

7.5 Gotu Kola (*Centella asiatica*)

Centella asiatica promotes cognitive function, memory, and concentration. It exhibits neuroprotective, antioxidant, and anti-inflammatory effects, making it valuable in the context of neurodegenerative disorders [31]. The therapeutic potential of these herbs in neurodegenerative disease management is evident, as natural compounds can target oxidative stress, inflammation, and neuronal damage, which are central to disease pathology. However, due to limited clinical investigations and restricted dosing studies, further research is necessary to fully elucidate their mechanisms, efficacy, and safety. Current evidence supports their use as complementary therapeutic approaches alongside conventional treatments[32].

8. *Tinospora cordifolia* as an Herb

Tinospora cordifolia, commonly known as Guduchi, Giloy, or Amrita, and referred to in English as heart-leaved moonseed, is a widely distributed evergreen climbing shrub native to India and found in tropical regions up to 1.2 km above sea level. It also grows in neighboring countries, including China, Sri Lanka, Bangladesh, Pakistan, and Myanmar [33]. The plant thrives in warm climates and prefers medium-black or red soils, but it can grow in a variety of soils—loamy, sandy clay loam, or clay loam—provided they are well-drained, moist, and rich in organic matter [34]. Traditionally considered divine in origin and referred to as the "food of God Indra", *T. cordifolia* has been highly valued for its medicinal properties. It is one of the most extensively researched and widely used traditional medicinal plants, with documented benefits in treating heart disease, diabetes, leprosy, rheumatoid arthritis, allergies, and other ailments [34-35].

8.1 Morphology

T. cordifolia is a climbing shrub with multiple coiling branches. The plant is divided into distinct parts, including stems, leaves, flowers, fruits, aerial roots, lamina, and seeds. Notable features of the aerial roots include tetra- to penta-arch structures, with a cortical layer composed of an outer thick wall and an inner parenchymatous zone. The lamina is typically ovate, ranging from 10–20 cm in length and up to 15 cm in

width, and is basal, subcordate, membranaceous, pubescent to sericeous, with a whitish tomentulose exterior and a strong granular reticulate structure beneath [36] (**Figure 1**).

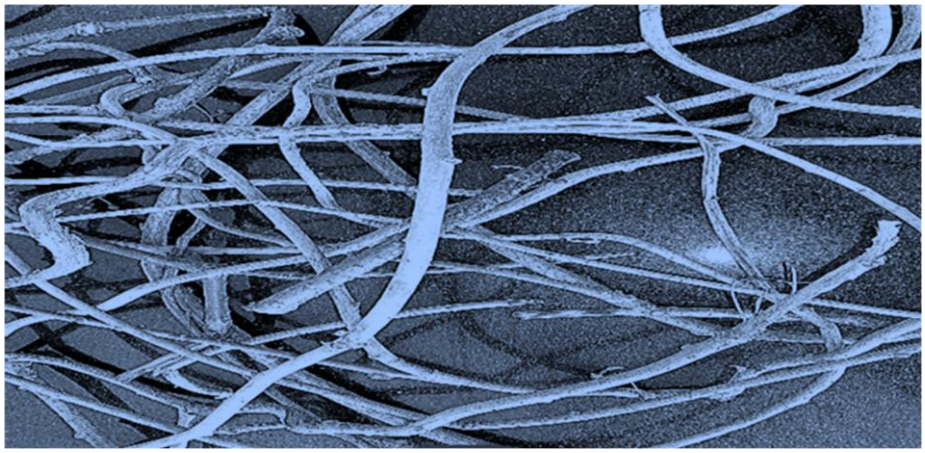

Figure 1: Stems of *Tinospora cordifolia*.

T. cordifolia, often regarded as the "queen of all herbs", holds significant potential for the treatment of numerous diseases and continues to be a focus of modern pharmacological research.

9. Cultural and Ethno-Pharmacological Significance of *T. cordifolia*

Tinospora cordifolia (Giloy) holds significant cultural and ethno-pharmacological value across various traditions, with particular importance in the Indian system of medicine. In Ayurveda, it is highly regarded for its versatility and health-promoting properties and is traditionally known as "Amrita", a Sanskrit term meaning the "root of immortality" [37]. Its historical applications in both Ayurvedic and allopathic medicine are summarized in **Table 2**.

Table 2: Historical Use of *Tinospora Cordifolia* in Ayurveda and Allopathy.

Aspect	Ayurveda	Allopathy	References
Traditional Uses	Used as a rejuvenator, immunomodulator, anti-inflammatory, and antipyretic	Limited historical use, primarily researched for its immunomodulatory and antipyretic properties	[38]
Forms of Administration	Decoction, powder, juice, tablets, and tinctures	Extracts and supplements, mainly in tablet or capsule form	[39]
Key Benefits	Enhances immunity, treats chronic fever, diabetes, jaundice, and arthritis	Supports immune function, reduces fever, and potential adjunct to diabetes management	[40]
Mechanism of Action	Believed to balance the three doshas (Vata, Pitta, Kapha), detoxify the body	Research indicates immunomodulatory, anti-inflammatory, and antioxidant properties	[33]
Clinical Studies	Extensive traditional use with some modern studies supporting efficacy	Fewer historical references, but modern studies validate certain benefits	[41]
Safety Profile	Generally considered safe, used for centuries in various formulations	Generally considered safe in recommended doses, with ongoing studies on long-term effects	[42]

10. Traditional Knowledge of Giloy

10.1 Ayurvedic Medicine

In Ayurveda, every part of Giloy has been used for its diverse therapeutic properties, including adaptogenic, anti-inflammatory, antipyretic, and immunomodulatory effects. It is traditionally employed to treat fevers, enhance immunity, and promote overall well-being [33].

10.2 Traditional Remedies

Historically, Giloy has been a cornerstone in natural remedies within Ayurvedic practice. Its antipyretic and antimicrobial activities have made it effective in managing chronic fevers and various infections [43]. Additionally, it supports gastrointestinal health, alleviating conditions such as indigestion, constipation, acidity, gas, and dyspepsia. Giloy is also recommended for respiratory disorders like asthma and chronic cough, highlighting its role in enhancing overall physiological performance [33].

10.3 Ritual and Cultural

In Hindu traditions, Giloy is revered as a divine plant, often associated with immortality and life-giving properties. It is believed to have spiritual importance and is considered protective and curative, reflecting its sacred status across various cultural practices [44].

10.4 Modern Applications

Modern research has emphasized Giloy's potential as an immune-boosting agent, a relevance that surged during the COVID-19 pandemic. Today, Giloy is widely available in forms such as tablets, powders, and juices, making it accessible for integration into contemporary healthcare practices [45].

11. Phytochemistry of *Tinospora cordifolia*

Tinospora cordifolia (Guduchi/Giloy) is rich in bioactive phytochemicals that contribute to its medicinal properties. The plant contains alkaloids, glycosides, steroids, diterpenoid lactones, sesquiterpenoids, phenolics, and polysaccharides, among others. These constituents are largely responsible for its therapeutic effects in traditional and modern medicine. Some of the key chemical compounds are summarized in **Table 3** [46].

Table 3: Chemical Constituents of *Tinospora cordifolia*.

S. No.	Compound Class	Compounds Identified	References
1	Alkaloids	Berberine, palmatine, magnoflorine, choline, tinosporin, jatrorrhizine	[47]
2.	Diterpenoid Lactones	Tinocordifolin, tinocordiside	[48]
3.	Glycosides	Tinosporaside, cordifolide, giloin, cordifolioside A, syringin	[49]
4.	Polysaccharides	Beta-sitosterol glucoside	[50]
5.	Phenolics	Arabinogalactan, glucans, catechins, tannins, gallic acid	[51]
6.	Essential Oils	Quercetin, kaempferol, rutin, caryophyllene, sesquiterpenoids, limonene, linalool	[52]

12. Pharmacology of *Tinospora cordifolia*

Tinospora cordifolia (Giloy) exhibits a wide range of pharmacological activities, which have made it one of the most extensively used medicinal plants [53]. A summary of its notable pharmacological properties is presented in **Table 4**.

Table 4: Pharmacological Properties of *Tinospora cordifolia.*

Property	Mechanism of Action	Reference
Anti-inflammatory	Inhibits pro-inflammatory cytokines (TNF-α, IL-1β, IL-6) and COX-2 enzyme	[54]
Immunomodulatory	Enhances macrophage activation, stimulates cytokine production	[55]

Antioxidant	Scavenging free radicals enhances endogenous antioxidant enzyme levels	[56]
Neuroprotective	Reduces oxidative stress, improves cognitive function, and inhibits neuronal apoptosis	[57]
Antidiabetic	Modulates insulin secretion and improves glucose metabolism	[58]
Hepatoprotective	Reduces liver enzyme levels, prevents hepatic fibrosis	[59]
Antimicrobial	Inhibits the growth of various bacteria and fungi	[60]
Anticancer	Induces apoptosis in cancer cells, inhibits tumor growth	[61]
Antipyretic	Reduces fever by inhibiting prostaglandin synthesis	[62]

13. Published Medical Research on Neuroprotection by Giloy (*Tinospora cordifolia*)

Extensive research has explored the neuroprotective properties of *Tinospora cordifolia* (Giloy) for the prevention and management of neurological disorders. Key findings from preclinical and clinical studies are summarized below:

13.1 Cognitive Enhancement and Alzheimer's Disease

Studies indicate that Giloy improves memory and cognitive function in models of Alzheimer's disease, primarily due to its antioxidant and anti-inflammatory effects [63].

13.2 Parkinson's Disease

In Parkinsonian rat models, Giloy extract suppressed neurodegeneration and improved motor function, likely through antioxidant-mediated neuroprotection [64].

13.3 Stress and Anxiety

Animal studies suggest that Giloy exhibits anxiolytic and antidepressant effects, reducing stress-related behaviors and supporting its use as an adaptogen [65].

13.4 Neuroinflammation

Giloy has demonstrated anti-inflammatory and neuroprotective effects in models of neuroinflammation, indicating its potential in mitigating inflammation-driven neuronal damage [33].

14. MOA of *Tinospora cordifolia* on neurodegenerative diseases

Giloy exerts neuroprotective effects via multiple mechanisms, including antioxidant, anti-inflammatory, anti-apoptotic actions, modulation of neurotransmitters, mitochondrial protection, and enhancement of neurotrophic factors.

14.1 Antioxidant Activity

Rich in phenolics, flavonoids, and terpenoids, Giloy combats oxidative stress, a key factor in neuronal injury. It also enhances endogenous antioxidant enzymes such as SOD, catalase, and GSH-Px, thereby strengthening neuronal defense against free radicals and supporting overall neuronal health [34].

14.2 Anti-inflammatory Effects

Giloy reduces neuroinflammation by inhibiting pro-inflammatory cytokines like TNF-α, IL-1β, and IL-6, and by suppressing the NF-κB signaling pathway, which limits the production of inflammatory mediators and protects neurons [66].

14.3 Anti-apoptotic Properties

Giloy modulates apoptosis-related genes, upregulating Bcl-2 (anti-apoptotic) while inhibiting Bax and caspases (pro-apoptotic), preventing neuronal cell death and supporting neuronal survival in neurodegenerative conditions [67].

14.4 Neurotransmitter Modulation

Giloy enhances dopamine levels and protects dopaminergic neurons in Parkinson's disease, while inhibiting acetylcholinesterase, increasing acetylcholine levels in Alzheimer's models, thereby improving motor and cognitive functions [68].

14.5 Mitochondrial Protection

Giloy preserves mitochondrial membrane integrity, preventing mitochondrial dysfunction—a key factor in neuronal degeneration—and supporting neuronal energy metabolism and survival [69].

14.6 Neurotrophic Factors

Giloy upregulates BDNF, promoting neurogenesis, neuronal differentiation, survival, and synaptic plasticity, which enhances cognitive function and brain resilience [70].

15 Specific Aspects Related to Neurodegenerative Disorders

15.1 Alzheimer's Disease (AD)

Giloy targets multiple AD pathophysiological processes. Its anti-amyloid effects help reduce beta-amyloid plaques, while its modulation of tau phosphorylation prevents neurofibrillary tangle formation, collectively slowing disease progression and improving cognitive outcomes [71].

15.2 Parkinson's Disease (PD):

15.2.1 Dopaminergic Neuron Protection

Giloy prevents dopaminergic neuron loss, improving motor symptoms such as tremors, rigidity, and bradykinesia.

15.2.2 α-Synuclein Aggregation

It inhibits the formation of α-synuclein aggregates, reducing neuronal toxicity and potentially slowing disease progression [72].

16 Conclusion

Tinospora cordifolia (Guduchi/Giloy) exhibits multifaceted neuropharmacological activities attributed to its rich phytochemical composition, including alkaloids, glycosides, steroids, and polysaccharides. Its antioxidant properties reduce oxidative stress, protecting neurons from free radical-mediated damage. Simultaneously, its anti-inflammatory effects suppress pro-inflammatory cytokines and modulate microglial activity, mitigating chronic neuroinflammation in disorders such as Alzheimer's and Parkinson's diseases. By modulating neurotransmitters, preventing apoptosis, protecting mitochondria, and enhancing neurotrophic factors, Giloy supports neuronal survival, synaptic plasticity, and cognitive function. These findings highlight the therapeutic potential of *T. cordifolia* as a natural agent against neurodegeneration, although systematic clinical trials are required to determine effective dosages, safety, and inclusion in therapeutic regimens. Its broad-spectrum mechanisms make it a promising candidate for future development of neuroprotective therapies.

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