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Therapeutic Potential of Rhododendron Arboreum: Phytochemical Constituents, Antioxidant Activity, and Neuroprotective Perspectives

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Abstract: *Neurodegenerative disorders, including Alzheimer's disease and Parkinson's disease, are major health challenges characterized by progressive neuronal dysfunction and cognitive decline. Oxidative stress, mitochondrial impairment, and excessive production of reactive oxygen species play a crucial role in the pathogenesis of these disorders. In recent years, medicinal plants rich in natural antioxidants have gained increasing attention for their potential neuroprotective benefits. Rhododendron arboreum, an important Himalayan medicinal plant, possesses a wide range of bioactive phytoconstituents such as flavonoids, phenolic compounds, quercetin, rutin, anthocyanins, and ascorbic acid, which contribute significantly to its therapeutic properties. This review highlights the phytochemical composition, antioxidant activity, and neuroprotective relevance of Rhododendron arboreum flower extracts. Various experimental studies have demonstrated strong free radical scavenging activity through assays such as DPPH, ABTS, FRAP, and hydrogen peroxide scavenging methods. These antioxidant properties may help protect neuronal cells from oxidative damage, reduce neuroinflammation, and enhance endogenous cellular defense mechanisms. Additionally, the plant exhibits several pharmacological activities including anti-inflammatory, cardioprotective, antimicrobial, hepatoprotective, and wound healing effects, supporting its medicinal importance. Overall, the available scientific evidence suggests that Rhododendron arboreum holds promising potential as a natural source of neuro protective agents. However, further in vivo investigations, mechanistic studies, and clinical evaluations are required to establish its safety, efficacy, and therapeutic application in neurodegenerative disorders.*

Keywords: Rhododendron arboreum; Neurodegeneration; Antioxidant activity; Phytochemicals; Oxidative stress; Neuroprotection; Flavonoids; DPPH assay; Alzheimer's disease; Parkinson's disease

I. INTRODUCTION

As neurons are crucial for communication, they are essential to the healthy operation of the human brain. Although neurons are found throughout the body, the majority of them start in the brain. Most neurons are produced by neural stem cells throughout childhood, but as people age, their numbers drastically decrease. Dysfunction of synapses, neuronal networks, and the accumulation of physiochemically modified protein variations in the brain are all linked to neurodegeneration. Neurodegenerative diseases are collectively referred to as NDs.1 It causes damage to the neurones

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or death of neurons in the central nervous system and which can lead to severe impairment like death. They are most frequently seen among senior citizens. On the other hand, illness could manifest earlier. They have become much more common in recent years, and as people age, this tendency is predicted to continue.2 About 1011–1012 neurons make up the adult brain, and at least twice as many neuroglial cells nourish and shield these neurons. Although critical molecules like glucose and the majority of lipid-soluble molecules may still get through, the endothelium of the brain's small blood vessels is far less permeable to molecules than other vascular endothelia. This so-called blood brain barrier (BBB) keeps many other chemicals out of the brain. In addition, circulating phagocytes remain out of a healthy brain by the BBB. Although the disease typically Characterized by genetic hallmarks, it is unknown if these hallmarks are the cause of the illness or if they are the result of other root causes. The most prevalent neurodegenerative disorders include Parkinson's disease, AD, and frontotemporal lobar degeneration (FTLD). Co-pathologies, or overlapping disorders, are common and contribute intricately to clinical presentations.3 Alzheimer disease (AD) is a progressive and irreversible dementia scenario marked by behavioural changes, loss of intellectual capability span several domains, incapacity to take care of oneself, and eventual problems in the nervous system. Neuritis plaques (containing amyloid proteins) and neurofibrillary tangles (paired, helical filaments containing tau proteins) grow in the cortex and hippocampus as a result of a series of events that cause AD. Age, head trauma, relatives who have a history of AD or Down syndrome, sex (more prevalent in women), and cardiovascular conditions are risk factors for the illness.4 Slowed movement (bradykinesia), resting tremor, muscular stiffness, and frequently postural instability are the hallmarks of Parkinson disease (PD) The initial clinical signs are usually solitary and may include limb dystonia and other aberrant movements such postural or action tremor. Fatigue, constipation, hyposmia, rapid eye motion (REM) sleeping patterns disorder, sadness, anxiety, and insomnia are common non-motor symptoms.5 Research indicates that the brain is in a state of dynamic equilibrium, just like many other tissues. It possesses an endogenous population of stem cells that may replenish cells lost in certain experimental lesions and multiply in response to pharmacological and environmental changes.[6]

II. ROLE OF OXIDATIVE STRESS IN NEURONAL DAMAGE:

Oxidative stress has long been recognised as a potential contributor to the development of some late-onset neurodegenerative disorders. The majority of aging speculation are based on the notion that oxidative damage, mitochondrial malfunction, and mutations are caused by accumulated oxidative stress. However, a number of disputes start to surface regarding this subject as the function of ROS in aging and illnesses related to age is more acknowledged.Many neurodegenerative illnesses, such as AD, PD, Huntington's disease, ALS, PSP, Friedreich's ataxia, neurodegeneration with brain iron accumulation and optic atrophy, display mitochondrial pathology. Respiratory chain malfunction and oxidative stress, decreased ATP generation, calcium dysregulation, mitochondrial permeability transition pore opening, disruptions in mitochondrial dynamics, and dysregulated mitochondrial clearance represent just a few examples of a broad spectrum of mitochondrial failure.7 It is commonly known that apoptosis contributes significantly to cell loss in neurodegenerative diseases including Alzheimer's, Parkinson's, and stroke. Furthermore, cellular damage during these neurodegenerative illnesses has been linked to reactive oxygen species (ROS). These ROS have the ability to oxidize biological macromolecules, which can lead to necrosis or apoptosis in the cells.8

III. EPIDEMIOLOGY:

In recent decades, the epidemiology of neurodegenerative conditions has mostly relied on descriptive research using clinical criteria. However, clinical definition is essentially insufficient for both early diagnosis and thorough characterization of various phenotypes. Given the necessity for an early diagnosis of the disease and a formulation of standard clinical and scientific diagnostic criteria, descriptive epidemiology requires a new framework to upgrade the profession of neurodegenerative research.9 Certain proteins (PrP, TDP-43, SOD1, α-synuclein, Aβ, tau, Langerhans islet peptide, and transthyretin) or recognized combinations of these may cause a systematic epidemiologic pattern. Other organ-limited illnesses linked to amyloid deposits may be included in new biologic models for CNDDs.10

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Neurodegenerative disorders are brought on by a loss of function in the peripheral nervous system and brain's nerve cells.11

Sign and Symptoms:

Neurodegenerative conditions and multiple kinds of psychopathology frequently lead to disruptions in mental, emotional, and social behaviour. Neurodegenerative disorders may offer a window into the neurological causes of these frequent symptoms since they exhibit considerably more distinct patterns of shrinkage in the brain.12 Many patients with dementia and cognitive decline brought on by Alzheimer's disease and related conditions like Parkinson's disease, Lewy body disease, vascular dementia, and frontotemporal degeneration, among others, experience depressive symptoms to varying degrees.13

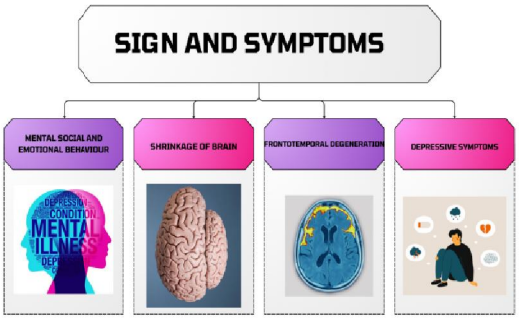

Figure 1: Sign and Symptoms of neurodegeneration.

Risk Factors:

For the majority of neurodegenerative disorders, such as Parkinson's disease (PD) and Alzheimer's disease (AD), aging is the main risk factor. AD affects 10% of those over 65, and its prevalence rises with age.14 While many neurodegenerative illnesses are growing connected to neuroinflammation, it is unclear if this is a cause or an effect of the disease process. The function of microbial infections in triggering degenerative neuroinflammatory responses and the genetic variables that may control such responses are of increasing interest.15 Gender, a low level of education hormonal issues, oxidative stress, inflammation, stroke, hypertension, diabetes, smoking, head trauma, depression, infection, malignancies, vitamin deficiencies, immunological and metabolic disorders, and chemical exposure are other potential factors.16 In addition, neurological diseases and memory losses may be correlated with inflammation and damage to vessels driven on by bacterial infections in the mouth.17

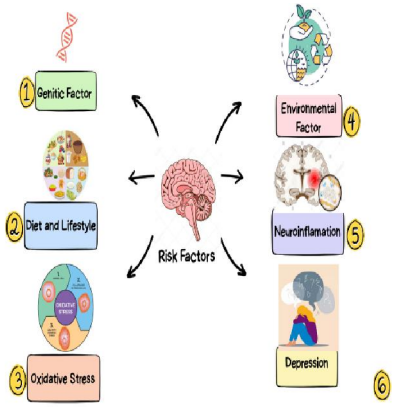

Figure 2: Risk Factors.

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Pathogenesis and Pathophysiology:

A formation of misfolded proteins, oxidative stress, neuroinflammation, excitotoxicity, and hereditary susceptibility are some of the causes of neurodegenerative diseases. Reactive oxygen species (ROS), which promote lipid peroxidation, DNA damage, and neuroinflammation, are produced more often when oxidative stress is present.18 Numerous lines of evidence point to a close relationship between AT defect, axonopathy, and neuroinflammation. It's yet undetermined, still, if these deficiencies are a direct cause of NDs or just an outcome of neuronal degeneration. Although a number of data indicate that these pathogenic pathways may begin early in ND pathophysiology, we currently lack trustworthy information on the temporal link between them.19

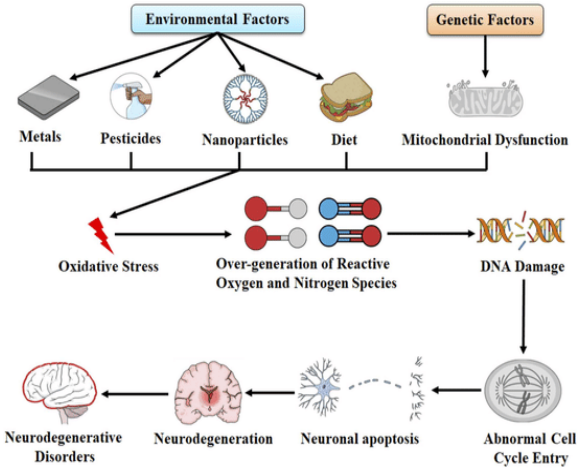

Figure 3: Pathophysiology of Neurodegeneration.

Importance of Antioxidant in Neuro protection:

Antioxidants have two modes of action: First, the antioxidant defence mechanisms, which are triggered in response to oxidative damage, stop the production of ROS and trap and block the radicals that are produced.20 An enzymatic antioxidant system consisting of glutathione peroxidase, catalase, and superoxide dismutase has been identified as the first line of defence. Reduced thiol and non-enzymatic antioxidants, such as hydro- and lipo-soluble or metabolic drugs, act as the second line of defence.21 Antioxidants work inside the body to stop free radicals from reacting with biological substances, stop the radical oxidation process, or inactivate the products of free radicals by fixing structural damage.22 As the understanding of the processes behind neuronal cell death during oxidative stress grows, it will be crucial to understand how antioxidants might disrupt signal transduction pathways and alter genetic programming. The encouraging studies on the neuroprotective effect of antioxidants in vitro and in vivo highlight a vital function for antioxidative active agents for the prevention and treatment of oxidative stress-related disorders, including AD, as long as specific therapies are unavailable.23

Medicinal Plant as Source of Neuroprotective Phytochemicals:

Throughout history, plant extracts have been utilized to treat an extensive range of diseases because of their wide variety of medicinal uses. But more recently, researchers have started looking at the biological functions of therapeutic plants, especially their neuroprotective effects.24 Based on numerous studies, natural chemicals like alkaloids and polyphenolic compounds that are extracted from plants might be capable to improve memory and cognitive function while also delaying neurodegeneration. Studies in ethnopharmacology as have provided information on potentially new medicines produced by plants.25 Numerous natural compounds, mostly plant extracts, have been used in traditional medicine for neuroprotective, memory-enhancing, and anti-aging purposes. These include Salvia officinalis, Bacopa monnieri, Curcuma longa, Panax ginseng, and Ginkgo biloba. These plants were investigated to determine whether the

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conventional assertion was accurate and to get further insight into the process by which they provide neuroprotective benefits.26

Rhododendron Arboreum:

Bright red flowers decorate the evergreen shrub or small tree Rhododendron arboreum. The Greek words "RHODO" (rose) and "DENDRON" (tree) are the source of the name "RHODODENDRON."The national flower of Nepal, rhododendron, is referred to as "Laligurans."27 Rhododendron arboreum is an important plant species both commercially and medicinally. It is widely recognized for its processed flower juice, which is now popular on the market as rhodojuice.28 The tincture of dried Rhododendron arboreum leaves has been used in homeopathic medicine to treat gout as well as astringent, diuretic, choleretic, antispasmodic, chronic eczema, diarrhoea, and dysentery.29 The woody members of the family Ericaceae, which are often used in a variety of ethno-medical uses, contain plants in the genus Rhododendron. It has approximately 1025 species in the genus Rhododendron, most of which are found at higher altitudes.30 Arunachal Pradesh, Jammu and Kashmir, Manipur, Meghalaya, Mizoram, Nagaland, Sikkim, Uttarakhand, West Bengal, and Himachal Pradesh are among the states in India where the plant is found.1 Numerous phytoconstituents, like ursolic acid, lupeol, sitosterol, taraxerol, and betulin, are present in the plant.For finding and refining a crude drug, the pharmacogenetic evaluation variables are essential.32

Figure 4: Rhododendron Arboreum.

Taxonomical Classification:

Kingdom.	*Plantae.*
Sub-kingdom.	*Tracheobionta.*
Super division.	*Spermatophyta.*
Division.	*Magnoliophyta.*
Class.	*Magnoliopsida.*
Order.	*Ericales.*
Family.	*Ericaceae.*
Genus.	*Rhododendron.*
Species.	*Rhododendron arboreum.*

Table 1: Taxonomical classification of Rhododendron Arboreum.33

Morphological Classification:

Character.	*R. Arboreum (Red Flower).*
Bark.	*Pinkish Brown.*
Leaf Shape.	*Oblong-lanceolate.*
Leaf Length.	*7.5-15cm.*
Flower Length.	*9cm.*
Flowers in each cluster.	*18-20.*
Cylax Shape.	*Triangular to Ovulate.*
Cylax Lobes.	*5.*

Table 2: Morphological Classification of Rhododendron Arboreum.34

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Pharmacological Properties:

Hepatoprotective activity:

The ethyl acetate and ethanolic isolates of R. arboreum have quercetin-related phenolic, saponin, and flavonoid components that have hepatoprotective effects.35

Anti-Diabetic activity:

The flowering of Rhododendron arboreum Sm was evaluated for anti-diabetic properties, and active chemicals were extracted. Rat intestinal α-glucosidase has been reported to be reduced by an aqueous methanolic extract of Laligurans flowers.36

Wound Healing activity:

Rhododendron Arboreum extracts exhibited suppression of protein denaturation. When compared to control groups, the combined extracts considerably improved wound healing in vitro, with greater doses speeding up closure rates.37

Cardio Protective effect:

Rhododendron arborium Sm flowers' ethanolic extract and n-butanol fraction both have beneficial cardioprotective effects.38

Anti Cancar activity:

There have been reports of rutin in the leaves. These elements may be the reason of the extracts' promising anticancer properties, shown by the SRB and crown gall tumour tests.39

Anti-Microbial potential:

The well diffusion method was used to assess the plant extract's antimicrobial properties. Plates were incubated at 35°C for a 24-hour period after plant extracts were allowed to diffuse in Muller-Hinton agar medium that was already seeded with the test organism.40

Anti-Oxidant activity:

Rhododendron arboreum leaves are a natural source of antioxidants that may be able to treat disorders linked to oxidative stress. Methanol and ethanol extracts showed strong antioxidant activity and were high in phenol and flavonoid levels.41 The anti-oxidant activity of ethanolic extract of Rhododendron Arboreum flower is also determined by DPPH assay.42

Phytochemical Screening:

There are a bunch of phytochemicals that have been found in this plant. Beta-amyrin, heptadecane, 22-stigmasten3-one, tetradecane, methyl ester, linoleic, linoleyl alcohol, beta-citronellol, dodecane, L-ascorbic acid, 2, 6-dihexadecanoate, alpha-amyrin, and dibutyl phthalate were the primary compounds found in the GC-MS analysis of methanolic leaf extract.43 Gossypetin, kempferol, azaleatin, caryatin, quercetin, and leucoanthocyanidin were the phenolic chemicals that Harborne and Williams isolated from alcoholic leaf extracts in 1971 using two-dimensional chromatography.44 The presence of coumaric acid, rutin, and quercetin in methanolic floral extract.45

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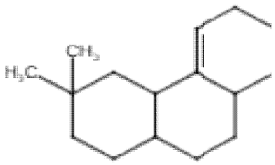
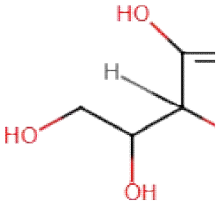
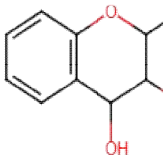
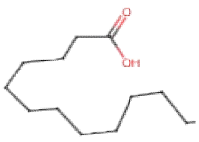
Chemical Structure.	Part of Plant.
Beta-amyrin	
	Leaf
L ascorbic acid	
	Leaf
leucoanthocyanidin	
	Leaf
Octadecanoic acid	
	Flower
Eicosane	

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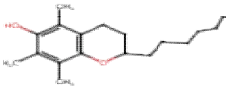
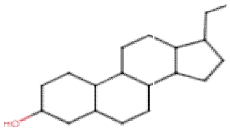
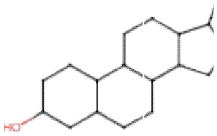
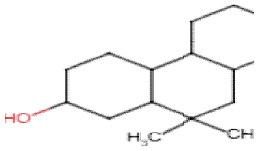
	Flower
Vitamin E	
	Flower
Stigmasterol	
	Flower
Campasterol	
	Flower
Taraxerol	
	Bark

Table 3: Phytochemical Screening.46,47

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Anti-oxidant activity of Rhododendron Arboreum:

As per some studies, the extracts were evaluated for total phenolics (123.6 mg GAE/g), anthocyanin content (1.76% w/w), and antioxidant qualities against assays for 2,2-diphenyl-1-picrylhydrazyl radical (IC50: 102.06 and 96.92 μg/mL) and 2,2′ -azino-bis (3-ethylbenzothiazoline-6-sulfonic acid) radical cation (112.25 and 45.59 μM TE/g).48 The 2, 2-diphenyl-1-picryl hydrazyl (DPPH) test was used to measure the antioxidant activity of the methanol extracts from various R. arboreum parts, and the radical scavenging activity (IC 50) was computed. Leaves had the greatest free radical scavenging action (IC 50 = 8.34 μg/ml), whereas stems had the lowest (IC 50 = 67.83 μg/ml).49 In analyses for DPPH, ABTS, hydrogen peroxide removal, lipid peroxidation inhibition, and FRAP, flower extracts revealed exceptional antioxidant activity. Due to the antioxidant activity of plant extracts, the study identifies an array of phytochemicals in R. arboreum extracts and highlights their potential uses in drugs, nutraceuticals, and functional foods.50

Antioxidant-mediated Neuroprotective Potential:

Neuroprotection has been extensively researched as a therapy option for a number of problems of the central nervous system (CNS), such as trauma, stroke, and neurodegenerative diseases. Antioxidants have a propensity to block or delay apoptosis. Numerous studies have revealed that certain natural antioxidants may have therapeutic benefits against cerebral ischemia damage.51 Vitagenes, such as Hsp70, heme oxygenase-1, thioredoxin, and sirtuins, play a crucial part in the cellular processes that provide protection against oxidative stress. Antioxidant enzymes and transcription factors like Nrf-2 and NFκB are often involved in cellular signaling pathways and molecular processes that drive hormetic responses. Vitagenes help prevent damage caused by ROS, either alone or in combination.52

IV. CONCLUSION

In conclusion, Rhododendron arboreum emerges as a promising medicinal plant with significant antioxidant and neuroprotective potential. The presence of diverse phytoconstituents, particularly flavonoids and phenolic compounds, contributes to its strong free radical scavenging activity and ability to mitigate oxidative stress, a key factor in neurodegeneration. Experimental evidence supports its role in protecting neuronal cells through modulation of oxidative pathways and enhancement of endogenous antioxidant defenses. Although current findings are encouraging, most studies are limited to in vitro and preliminary investigations. Therefore, further in vivo studies and clinical trials are necessary to validate its efficacy, safety, and therapeutic applicability. The integration of R. arboreum into modern pharmacotherapy could offer a natural, cost-effective approach for the prevention and management of neurodegenerative disorders.

Ethics:

This study was a secondary analysis based on the currently existing data and did not directly involve with human participants or experimental animals. Therefore, the ethics approval was not required in this paper.

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Conflict of interest:

The author declares that there is no conflict of interest.

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Data Access:

The data that supports the finding of this study are available from the corresponding author upon reasonable individual request.

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