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A Review Article: Pharmacology of Phytochemicals in Neurodegenerative Disorders

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Abstract: ***Neurodegenerative disorders (NDs), including Alzheimer's disease, Parkinson's disease, Huntington's disease, and amyotrophic lateral sclerosis, are growing conditions characterized by neuronal loss, cognitive decline, and motor impairment. Current pharmacological treatments generally offer symptomatic relaxation and are insufficient to halt disease progress. Phytochemicals—bioactive compounds derived from plants such as polyphenols, flavonoids, alkaloids, terpenoids , and stilbenes—have emerged as promising candidates due to their multi-target therapeutic potential. The compounds exhibit antioxidant, anti-inflammatory, anti-amyloidogenic, and mitochondrial-protective activities, while further regulating neurotransmission and proteostasis. Prominent phytochemicals such as curcumin, resveratrol, EGCG, quercetin, ginsenosides , and huperzine A have explained neuroprotective effects in preclinical and early clinical studies Still, challenges such as poor solubility, poor bioavailability, fast metabolism, and lack of standardized formulations limit their clinical translation. Emerging planning , containing Nano carriers, liposomal systems, and solid lipid nanoparticles, offer enhanced delivery and brain targeting. Additionally, personalized medicine approaches, biomarker-based clinical trials, and combination therapies with conventional drugs may improve treatment outcomes. This review specifies an overview of the pharmacological mechanisms of key phytochemicals in NDs, evaluates current evidence, and highlights future prospects and challenges in their therapeutic growth. Advancing the compounds from bench to bedside requires rigorous clinical confirmation, optimized formulations, and regulatory standardization***

Keywords: ***Phytochemicals, Neurodegenerative Disorders, Neuroprotection, Oxidative Stress , Protein Aggregation , Bioavailability , Combination Therapy , Personalized Medicine***

I. INTRODUCTION

Neurodegenerative disorders such as Alzheimer's disease, Parkinson's disease, Huntington's disease, and related cognitive and motor decline conditions represent one of the most serious public health challenges of the twenty-first century. These disorders are characterized by gradual but irreversible neuronal loss, synaptic dysfunction, abnormal protein aggregation, mitochondrial damage and neuro inflammatory processes that progressively impair memory, movement, coordination, and behavior [1] As the global population continues to age, their incidence has raised significantly, adding substantial clinical, social, and financial burden across both developing and developed regions. Despite decades of research, now available therapeutic planning remain mainly symptomatic and fail to modify underlying disease mechanisms or halt progress. Normal pharmacotherapy, including cholinesterase inhibitors or dopaminergic agents, supplies only temporary symptomatic relaxation and is frequently associated with side effects or loss of efficacy over time [2]. The restraints underscore this urgent need for secure, multi-target therapeutic approaches capable of intervening in oxidative stress pathways, neuroinflammatory cascades, neurotransmitter dysregulation, and protein misfolding events this collectively drive neurodegeneration. In this circumstances, phytochemicals—naturally occurring bioactive compounds derived from plants—have emerged as promising alternatives and adjuvant candidates for neuroprotection. A growing body of evidence indicates that polyphenols, flavonoids, alkaloids, terpenoids, and other phytoconstituents can modulate several molecular targets simultaneously, thereby exerting antioxidant, anti-amyloid, anti-inflammatory, anti-apoptotic, and metal-chelating effects [3,4]. For instance, certain polyphenols have been reported to suppress microglial activation, reduce reactive oxygen species generation, inhibit acetylcholinesterase, and improve mitochondrial function, while others can interfere with β-amyloid aggregation or tau hyperphosphorylation [5]. Additionally, many phytochemicals demonstrate favorable interactions with basic indicating pathways the regulate neuronal survival, synaptic plasticity, and neurotrophic support. They can activate endogenous defense systems such as Nrf2-dependent antioxidant reactions and suppress pro-inflammatory mediators via NF-κB restriction, helping to restore redox balance and reduce chronic inflammation in the aging mind [4]. Additionally, few plant-derivative alkaloids and flavonoids influence cholinergic and dopaminergic neurotransmission, contributing to the maintenance of cognitive and motor function.

Experimental studies also highlight the capacity of herbal compounds until cross the blood–brain barrier under certain formulations, enabling direct safety of central nervous system cells. However, the translation of phytochemicals from preclinical findings to clinical effectiveness remains challenging. Issues such as poor bioavailability, low solubility, fast metabolism, and variability in plant sources can limit their pharmacological utility [3]. To address these constraints, recent advances have focused on improving delivery systems, optimizing extraction and purification methods, and combining multiple compounds to enhance synergistic action. This integration of historic medicinal information with new pharmacological approaches has also increased the discovery and evaluation of promising phytochemical candidates. Modern reviews emphasize that future research must focus on molecular mechanisms, dosage standardization, and long-term security confirmation to advance these agents into evidence-based therapeutic frameworks [1,5]. Furthermore, this growing interest in dietary interventions, functional foods, and nutraceuticals has reinforced that relevance of phytochemicals not only for treatment but further for prevention or delay of neurodegenerative onset. The equivalence between regular consumption of plant-based nutrients as well as reduced cognitive decline has drawn significant attention in both clinical and epidemiological studies, further supporting the investigation of organic compounds for brain health [2,4]. Considering the complex and multifactorial nature of neurodegenerative disorders, the multi-target and pleiotropic effects of phytochemicals offer a distinct advantage over single-target synthetic drugs. Their relative security profile, biocompatibility, and accessibility improve their potential as complementary or alternative interferences. Accordingly, exploring the pharmacology of phytochemicals in neurodegenerative disorders provides a realistic and timely direction for developing novel therapeutic planning that can act on multiple pathological pathways simultaneously. [3,4]

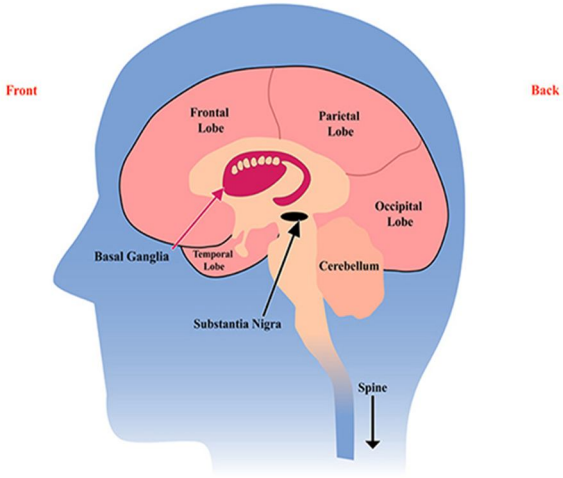

Fig no 1- Side view of the brain, sliced in half, showing the basal ganglia (red), and the substantia nigra [42]

II. TYPES OF NEURODEGENERATIVE DISORDERS

1) Alzheimer's Disorder (AD):- The most common neurodegenerative disorder, resulting from growing memory defeat, intelligent impairment, and concern with changes in manner of behaving. It guides abnormal accretion of amyloid-β plaques and tau tangles in the brain [6]
2) Parkinson's Disorder (PD) :- A change disorder caused by degeneration of dopaminergic neurones in the substantia nigra, superior to tremors, rigidity, and bradykinesia [7]
3) Huntington's Disease (HD):- An inherited disorder caused by CAG repeat growth in the HTT gene, resulting in motor dysfunction, psychiatric symptoms, and growing cognitive decline [8]
4) Amyotrophic Lateral Sclerosis (ALS):- A critical motor neurone disease dominant in muscle weakness, paralysis, and respiratory deficiency due to degeneration of motor neurones [9]
5) Motor Neurone Disease (MND):- A group of conditions, including ALS, from progressive damage of motor neurones, leading to muscle destruction and impaired motion [10]
6) Multiple System Atrophy (MSA): - A rare disorder accompanying features of Parkinsonism, cerebellar disorder, and autonomic dysfunction caused by extensive neuronal damage [7]
7) Frontotemporal Dementia (FTD): -A group of disorders generally affecting the frontal and temporal lobes, causing trait changes, disturbances concerned with manner of behaving, and language degradation [6,8]

8) Prion Diseases (e.g., Creutzfeldt–Jakob disease):- Unique, rapidly growing neurodegenerative disorders caused by misfolded prion proteins, superior to dementia, motor dysfunction, and death [11]

III. ETIOLOGY AND PATHOPHYSIOLOGY OF NEURODEGENERATIVE DISORDERS

Aetiology and Pathophysiology of Neurodegenerative Disorders Neurodegenerative disorders (NDs) are complex, growing conditions from gradual loss of neuronal structure and function, eventually resulting in cognitive decline, motor degradation, and premature death. Their aetiology (causative factors) and pathophysiology (mechanistic processes) are multifactorial, involving interaction between genetic predisposition, environmental uncoverings, ageing, and basic dysfunction

A. Etiological Factors

Ageing is the most powerful risk consideration for nearly all neurodegenerative disorders. With accelerating age, neurones accrue oxidative damage, injured DNA repair, mitochondrial dysfunction, and lowered proteostasis. Cellular clearance mechanisms like autophagy and proteasomal degradation also decline, making the mind more exposed to protein collection and stress [12,14]

Genetic Susceptibility Certain genetic variations predispose persons to NDs. For example, the APOE ε4 allele increases the risk of Alzheimer's disorder, while mutations in SNCA, LRRK2, and Parkin contribute to Parkinson's disease. Similarly, expansions of trinucleotide repeats in the HTT gene cause Huntington's disease. These genes usually influence pathways regulating protein management, mitochondrial function, and inflammatory reactions [12,13]

Environmental and Lifestyle Factors Chronic exposure to pesticides, heavy metals, or air pollution increases oxidative stress and mitochondrial harm in neurones. Similarly, metabolic disease, diabetes, and weak behaviour selections (smoking, inactivity, and a sick diet) create systemic stress that stimulates neurodegeneration. These exposures frequently communicate with genetic susceptibility, making some individuals more vulnerable than others [14]

Immune and Systemic Contributions Beyond the mind, peripheral immune dysregulation and chronic systemic inflammation influence the central nervous system. Cytokines and inflammatory mediators can cross the blood–brain barrier and turn on microglia, initiating long-term inflammatory cascades that enhance neuronal damage [12]

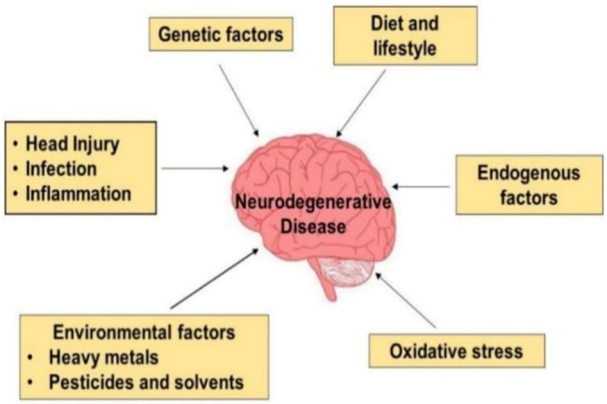

Fig no.2- Etiology of neurodegenerative diseases [43]

B. Pathophysiological Mechanisms

Protein Misfolding and Aggregation A symbol of most NDs is the build-up of misfolded proteins: amyloid-β and tau in Alzheimer's, α-synuclein in Parkinson's, huntingtin in Huntington's disease, and prion proteins in prion disorders. These proteins misfold into harmful conformations that oligomerize and aggregate, breaching cell membranes, impairing synapses, and triggering apoptosis [14]

Prion-like Propagation Recent evidence suggests that misfolded proteins can spread between cells in a prion-like category. For instance, α-synuclein and tau aggregates may be released from infected neurones, taken up by neighbouring cells, and act as templates for further misfolding. This describes why pathology progresses in predictable anatomical patterns over a period [14,15]

Mitochondrial Dysfunction and Energy Failure Neurones rely heavily on oxidative phosphorylation for ATP.

Dysfunction in mitochondrial composites leads to strength failure, injured calcium buffering, and raised ROS production. Mutations in mitochondrial DNA, as well as age-related decline in mitochondrial dynamics (fusion/fission), further compromise neuronal survival [13]

Oxidative and Nitrosative Stress Excessive reactive oxygen species (ROS) and reactive nitrogen species (RNS) damage lipids, proteins, and deoxyribonucleic acid. Antioxidant defence arrangements such as glutathione, catalase, and superoxide dismutase decline with age, tipping the balance toward oxidative damage, which stimulates collection and cell death [13]

Proteostasis Collapse Cellular protein quality control mechanisms, including the ubiquitin–proteasome structure and autophagy–lysosome path, become inefficient under chronic stress. This results in damaged clearance of broken proteins and organelles that grow and perpetuate toxicity [14]

Neuroinflammation Microglia and astrocytes put themselves in the place of other protein aggregates and neuronal stress by releasing cytokines, chemokines, and sensitive species. While initially protective, chronic activation enhances maladaptation, causing synaptic stripping, neuronal harm, and further propagation of pathology [12]

Excitotoxicity and Calcium Dysregulation Excessive glutamate release along with impaired reuptake leads to overactivation of NMDA receptors, calcium overload, and activation of apoptotic indicating cascades. Calcium inequality again exacerbates mitochondrial failure, forming a vicious cycle of excitotoxic damage [13,15]

Network-Level Propagation Molecular pathology at the cellular level translates into chain dysfunction. Abnormal protein assemblies, synaptic failure, and neuroinflammation spread across anatomically related brain areas, producing the progressive and stereotyped clinical syndromes noticed in Alzheimer's, Parkinson's, and other NDs [14,15]

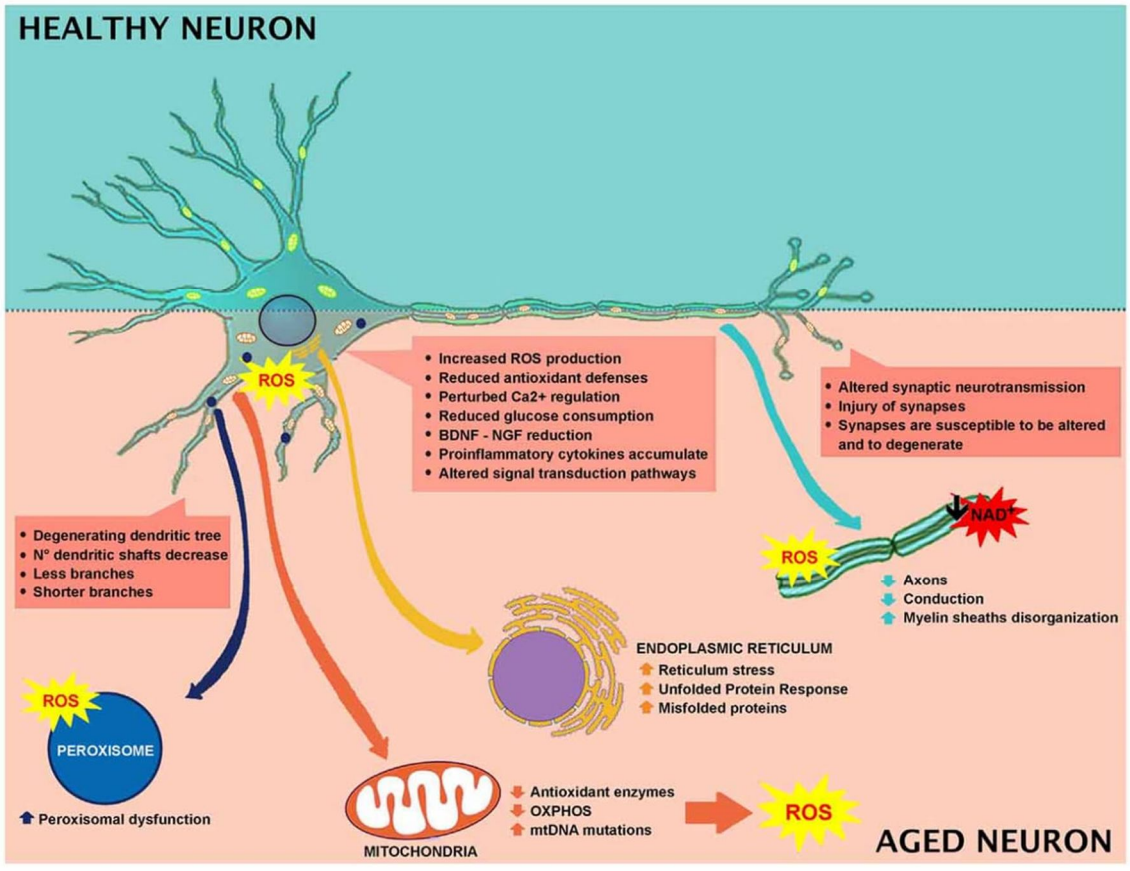

Fig 3 - Effect of aging in neuronal cell and involved mechanisms [44]

IV. DIAGNOSIS OF NEURODEGENERATIVE DISORDERS

Diagnosis of Neurodegenerative Disorders Diagnosis of neurodegenerative disorders combines clinical judgement, biomarkers, genetic testing, and advanced imaging methods. Accurate diagnosis guides patient administration, prognosis, and future therapy options.

1) Clinical and Neurological Assessment:- Initial diagnosis includes detailed history-taking, neurological tests, and assessment of cognitive and motor function. Tools like the Mini-Mental State Examination (MMSE) or Montreal Cognitive Assessment (MoCA) help screen cognitive impairment, while motor symptoms like tremors, severity, or gait changes suggest specific disorders like Parkinson's disease [16]
2) Imaging Techniques:- MRI and CT scans help exclude fundamental causes (such as tumours and strokes) and recognise characteristic patterns like hippocampal atrophy in Alzheimer's or basic ganglia changes in Parkinson's. PET scanning utilising tracers for amyloid or tau proteins provides in vivo imaging of pathological protein accumulations, growing demonstrative precision [17]
3) Fluid Biomarkers:- Cerebrospinal fluid (CSF) measurements of amyloid-β, total tau, and phosphorylated tau are informational for Alzheimer's disease diagnosis. Other fluids like plasma or saliva are under examination as less invasive options. New biomarkers (e.g., neurofilament light chain) show promise in monitoring progress across different NDs [18]
4) Genetic Testing:- Testing for mutations in known risk genes (APP, PSEN1/2 for Alzheimer's; SNCA, LRRK2 for Parkinson's; HTT for Huntington's) allows definitive diagnosis, particularly in familial and early-onset cases. Next-generation sequencing (NGS) allows broader genetic study and is revolutionising molecular diagnostics [19]
5) Emerging Approaches:- Digital and Robotic Biomarkers Innovative diagnostic procedures include robotic systems that analyse subtle motor and eye-activity patterns to notice early neurodegenerative changes noninvasively. These digital biomarkers enable objective calculations that may aid early discovery and tracking [16]

V. CURRENT MANAGEMENT APPROACHES

Current Management Approaches in Neurodegenerative Disorders Neurodegenerative disorders (NDs), in the way that Alzheimer's disorder (AD), Parkinson's disease (PD), Huntington's disease (HD), amyotrophic lateral sclerosis (ALS), and Niemann–Pick type C (NPC) are growing conditions from neuronal loss, protein misfolding, and impaired neurotransmission. As curative therapies are still not available, current administration focuses on alleviating symptoms, delaying disease progress, and developing quality of life through pharmacological and non-pharmacological approaches

A. *Pharmacological Interventions*

1) Symptomatic Therapies Alzheimer's disease (AD): - Cholinesterase inhibitors, in the way that donepezil, rivastigmine, and galantamine increase acetylcholine availability and enhance cognitive functions in mild-to-moderate AD. Memantine, an NMDA receptor antagonist, reduces excitotoxic neuronal injury in moderate-to-harsh stages [20,24]
2) Parkinson's disease (PD):- Levodopa remains the gold-standard therapy, replenishing dopamine and developing motor dysfunction. Dopamine agonists (e.g., pramipexole, ropinirole), MAO-B inhibitors (selegiline, rasagiline), and COMT inhibitors (entacapone) are generally used as adjuncts to optimise response and reduce motor vacillations Huntington's disease (HD): Tetrabenazine and deutetrabenazine, vesicular monoamine transporter (VMAT2) inhibitors, reduce hyperkinetic activities. Antipsychotics are still recommended for psychiatric manifestations Amyotrophic lateral sclerosis (ALS): Riluzole, which reduces glutamatergic excitotoxicity, and edaravone, an antioxidant, modestly slow disease progress and improve continuation [24]
3) Disease-Modifying Therapies Niemann–Pick type C (NPC):- Miglustat, a glycosphingolipid synthesis inhibitor, and 2-hydroxypropyl-β-cyclodextrin, a cholesterol-binding agent, show benefits in slowing neurological decline. These powers represent how targeted pharmacology can change disease trajectory in unique NDs [21]
4) Future directions: Disease-changing medicines under clinical trial involve monoclonal antibodies targeting amyloid-β (aducanumab, lecanemab) in AD and α-synuclein-supervised immunotherapies in PD. These novel powers show an example of a shift from symptomatic to mechanism-based therapy [24]
5) Adjunctive Neuropsychiatric Management NDs commonly present with indifference, depression, and anxiety. Stimulants, SSRIs, and dopaminergic drugs have been explored for these symptoms, though clinical results are mixed [22]

B. *Non-Pharmacological Approaches*

1) Physical Rehabilitation and Exercise :- Exercise-based interventions, containing aerobic preparation, resistance exercises, yoga, and tai chi, have been associated with upgraded motor coordination, mood, and overall range of capabilities. In PD, structured physiotherapy helps reduce gait disturbances and fall risk [23]

2) Cognitive and Behavioural Interventions :- Cognitive stimulation therapy (CST), reminiscence therapy, and memory preparation programmes enhance mental activity and delay decline in early dementia. Behavioural interventions still improve carer-patient interaction and reduce carer burden [23]
3) Lifestyle and Nutritional:- Modifications Dietary supplements like omega-3 fatty acids, antioxidants, and vitamins are commonly used to support mind health. Although clinical evidence varies, digestive designs are trusted to decrease oxidative stress and inflammation [21,23]. Additionally, maintaining regular sleep phases, social interaction, and organised routines are beneficial for long-term care [20]
4) Assistive Technologies and Digital Tools :- The use of robotics, wearable sensors, and telemedicine is extending. Robotic-assisted walk preparation boosts motor performance, while digital intelligent preparation platforms support thought and attention. These tools allow personalised rehabilitation and continuous disease monitoring [20]
5) Multidisciplinary and Holistic:- Care The best outcomes are achieved through comprehensive care models that merge neurologists, psychiatrists, physiotherapists, nutritionists, and carers. Multimodal therapy, combining drugs with behaviour interferences, is immediately considered the standard of care in many NDs [20,24]

VI. ROLE OF PHYTOCHEMICALS IN NEURODEGENERATIVE DISORDERS

Overview of Phytochemicals in Neurodegeneration:- Phytochemicals are naturally occurring bioactive substances present plentifully in fruits and vegetables, and growing regular consumption of such plant-based meals has been emphasised due to their potential health-promoting effects.

These compounds have been shown to offer protection against oxidative stress and neuroinflammation, both of which are major contributors to the progress of neurodegenerative disorders. Commonly encountered phytochemicals in everyday life involve curcumin, quercetin, flavonoids, epigallocatechin-3-gallate (EGCG), and diallyl trisulfide. In addition to their antioxidant actions, they have been associated with modulation of the immune system, regulation of hormone metabolism, and decline of platelet aggregation. Dietary patterns such as the Mediterranean diet, specifically rich in olive oil and plant-derivative compounds, have also been linked to decreased mortality in patients with conditions like Parkinson's and Alzheimer's disease. Although miscellaneous studies suggest that phytochemicals can function as nutraceuticals to alleviate neurodegenerative environments, the precise molecular mechanisms remain unclear.

It has been projected that their advantageous effects are mediated through radical scavenging and antioxidative activity, while additional hypotheses indicate involvement in stress-response activating, modulation of enzyme metabolism and gene expression, or interaction with cellular receptors to trigger downstream signalling pathways[3]

Neurodegenerative disorders like Alzheimer's disease, Parkinson's disease, and Huntington's disease are from progressive neuronal loss, oxidative stress, protein aggregates, and neuroinflammation. Conventional pharmacological approaches frequently provide only symptomatic relaxation, with limited capability to modify disease progress [1–3]. In this context, phytochemicals—bioactive compounds derived from plants—have attracted significant attention due to their neuroprotective properties, security characterisation, and wide availability. These compounds act through different mechanisms, such as antioxidant activity, modulation of neurotransmitters, anti-inflammatory effects, and stopping of protein misfolding and aggregation [26,27,3]

A. Classes of Phytochemicals and Their Role

1) Polyphenols

Polyphenols, containing phenolic acids, flavonoids, and stilbenes, are acknowledged for their effective antioxidant and anti-inflammatory effects. In neurodegenerative disorders, polyphenols such as curcumin, resveratrol, and epigallocatechin gallate (EGCG) restrict amyloid-beta aggregation, improve mitochondrial function, and decrease neuroinflammation [3,4]. Curcumin reduces oxidative stress markers and protects neuronal cells from degeneration, while resveratrol activates sirtuin pathways, enhancing neuronal survival [27]

2) Flavonoids

Flavonoids, a bigger subgroup of polyphenols, involve flavones, flavonols, flavanones, and anthocyanidins. Quercetin, kaempferol, and luteolin are notable examples with powerful neuroprotective effects. They modulate MAPK and PI3K/Akt pathways, suppress pro-inflammatory cytokines, and enhance synaptic plasticity [2,4]. Evidence suggests that flavonoid-rich diets correlate with reduced risk of cognitive decline and enhanced memory performance [3]

3) Alkaloids

Alkaloids are nitrogen-containing compounds with various pharmacological actions. Some act as acetylcholinesterase inhibitors; that is advantageous in Alzheimer's disease management. Galantamine, an alkaloid from the Galanthus class, was previously clinically approved for AD therapy. Huperzine A prevents glutamate-induced excitotoxicity and improves cognitive performance [25,27]

4) Terpenoids

Terpenoids, in the way that ginkgolides and bilobalide from Ginkgo biloba do, improve cerebral blood flow, protect neurones from apoptosis, and improve memory functions [4,26] Other terpenoids like asiatic acid and ursolic acid still exhibit antioxidant and anti-inflammatory features, countering neurodegenerative processes [3]

5) Saponins

Saponins, particularly ginsenosides from Panax ginseng, regulate neurotransmitter release, prevent neuroinflammation, and promote neuronal conversion. They again decrease amyloid-beta accumulation and tau hyperphosphorylation, suggesting their potential in Alzheimer's disease prevention [25,26]

6) *Carotenoids*

Carotenoids like β-carotene, lutein, and lycopene are a part of antioxidants by quenching reactive oxygen species and defending neuronal membranes from lipid peroxidation. Lutein and zeaxanthin have been connected to better cognitive efficiency and decreased age-related decline [27]

7) Stilbenes

Resveratrol, the most studied stilbene, activates sirtuin-1 (SIRT1), improves mitochondrial biogenesis, and exhibits anti-amyloidogenic properties. It too reduces neuroinflammation, making stilbenes strong applicants for slowing neurodegeneration [26,3]

B. Mechanisms of Neuroprotection

1) Phytochemicals act through multiple interconnected pathways:-
2) Antioxidant defence:- scavenging free radicals and improving antioxidant enzymes to a degree SOD and catalase [13,27]
3) Anti-inflammatory actions:- restriction of NF-κB and cytokine release [3,25]
4) Anti-amyloid activity:- preventing collection of amyloid-suspect and tau proteins [26]
5) Mitochondrial protection:- developing energy metabolism and reducing oxidative damage [4]
6) Metal chelation:- binding waste iron and copper ions, lowering metal-induced oxidative stress [27]

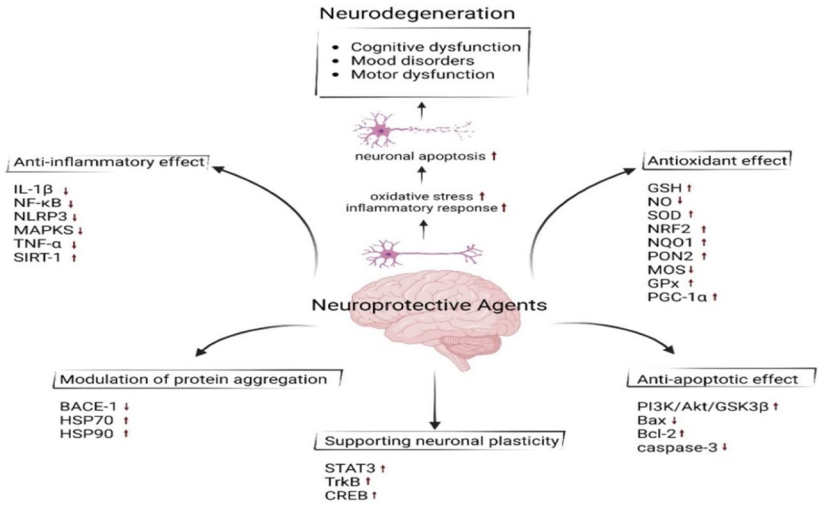

Fig 4 Mechanism of Neuroprotection [45]

VII. PHYTOCHEMICALS WITH PHARMACOLOGICAL SIGNIFICANCE

Phytochemicals represent a various group of naturally occurring bioactive compounds derived from plants that have shown promising pharmacological potential in the management of neurodegenerative disorders (NDs). Their neuroprotective properties are primarily mediated through antioxidant, anti-instigative, anti-apoptotic, and anti-amyloidogenic mechanisms that directly target the pathological hallmarks of disorders such as Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), and amyotrophic lateral sclerosis (ALS) [28,29,30]

Main categories of phytochemicals that have been widely studied include polyphenols, alkaloids, terpenoids, and flavonoids

A. Polyphenols

Polyphenols are among the most widely studied phytochemicals due to their potent antioxidant action and ability to modulate various indicating pathways relevant to neuronal survival

1) Curcumin :- A polyphenol derived from Curcuma longa, demonstrates strong free radical scavenging characteristics and inhibits lipid peroxidation. It can modulate basic factor erythroid 2–related factor 2 (Nrf2), thereby improving antioxidant defence, and simultaneously suppress nuclear factor-kappa B (NF-κB) signalling, decreasing neuroinflammation [28] Curcumin also restricts amyloid-β aggregation and promotes disaggregation of preformed fibrils, highlighting its therapeutic relevance in AD [29]
2) Resveratrol, present especially inwine and red wine, has been proved to activate sirtuin-1 (SIRT1), leading to improved mitochondrial biogenesis, reduced oxidative stress, and modulation of apoptotic pathways. In experimental PD models, resveratrol attenuates dopaminergic neuronal loss and improves autophagic clearance of toxic protein aggregates [30] Quercetin, a flavonoid-like polyphenol in the direction of apples and onions, is recognised for its powerful antioxidative and anti-inflammatory actions. Quercetin stabilises mitochondrial function, reduces microglial activation, and suppresses pro-inflammatory cytokines such as TNF-α and IL-1β. Additionally, it promotes neuronal survival by modulating the PI3K/Akt and MAPK pathways, which are critical for cell signalling and survival [28,31]

B. Alkaloids

Alkaloids are another main class of phytochemicals with neuropharmacological action.

1) Huperzine A, derived from Huperzia serrata, is a reversible acetylcholinesterase prevention with clinical importance in AD. It enhances cholinergic transmission by preventing acetylcholine breakdown, thereby improving thought and cognitive functions [32] Other than its symptomatic benefits, huperzine A again exhibits neuroprotective effects through anti-oxidative stress and anti-apoptotic properties.Galantamine, an alkaloid extracted from the Galanthus class, mostly functions as an acetylcholinesterase inhibitor but particularly acts as an allosteric modulator of nicotinic acetylcholine receptors. This double activity enhances synaptic pliancy and neurotransmission, making it a well-established drug for mild-to-moderate AD [32,33]
2) Terpenoids:- Terpenoids, specifically ginsenosides derived from Panax ginseng, have gained significant attention in neuroprotection.Ginsenosides exert multimodal actions, including antioxidant activity, mitochondrial protection, and inhibition of excitotoxicity interfered with by extra glutamate release. In PD models, ginsenosides reduce dopaminergic neuronal death by suppressing neuroinflammation and modulating neurotrophic factors in the way that brain-derived neurotrophic factor (BDNF) [33] does. Additionally, they increase synaptic plasticity and memory efficiency in AD models through modulation of cholinergic neurotransmission and anti-apoptotic effects [34]

C. Flavonoids

Flavonoids are abundant plant-derivative compounds with strong neuroprotective characteristics owing to their capability to cross the blood–brain barrier (BBB) and interact with neuronal signalling

1) Cascades. Epigallocatechin gallate (EGCG),:- The most active catechin in green tea, exhibits various neuroprotective mechanisms. EGCG scavenges reactive oxygen species (ROS), chelates transition metals, and suppresses amyloid fibril composition. It also inhibits α-synuclein aggregation, thus showing therapeutic potential in both AD and PD [29]
2) Kaempferol:- Widely present in vegetables and fruits, exerts antioxidant and anti-inflammatory actions. It reduces oxidative damage by upregulating Nrf2-related antioxidant enzymes and downregulating NF-κB–mediated inflammatory pathways has also been shown to prevent amyloid aggregation, save dopaminergic neurones, and modulate mitochondrial homeostasis, suggesting a broad neuroprotective role across differing NDs [28, 31,29]

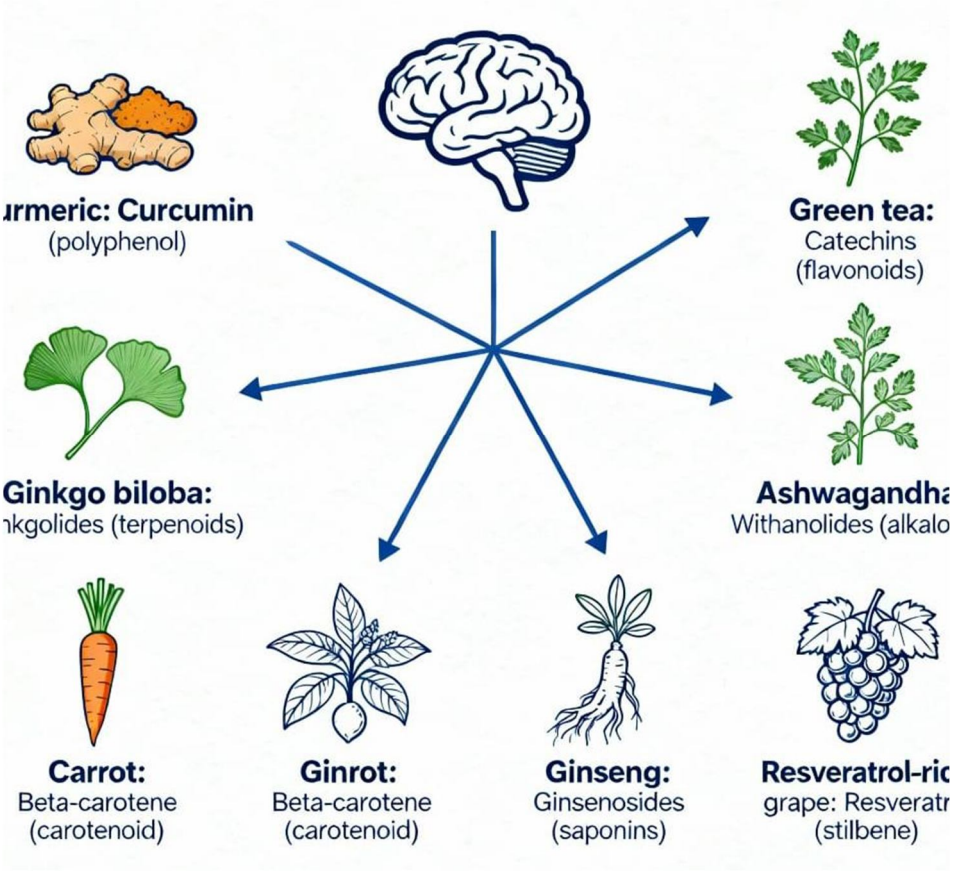

Fig 5- Plant with Bioactive Compounds for Neurodegenerative Disorders

VIII. CLINICAL EVIDENCE OF PHYTOCHEMICALS IN NEURODEGENERATIVE DISORDERS

Neurodegenerative disorders (NDs) such as Alzheimer's disease (AD), Parkinson's disease (PD), and additional forms of dementia remain major public health challenges due to their chronic progression, restricted curative therapies, and increasing predominance worldwide. In this context, phytochemicals have gained attention as potential therapeutic agents because of their antioxidant, anti-inflammatory, and neuroprotective characteristics. Clinical evidence, though still developing, provides valuable insights into their therapeutic potential and the challenges of translating preclinical findings into effective human treatments.

A. Curcumin in Alzheimer's Disease

Curcumin, a polyphenolic compound that comes from Curcuma longa, has been extensively studied for its potential to modulate oxidative stress, amyloid pathology, and neuroinflammation. Human clinical tests have evaluated curcumin supplementation in AD patients to assess its impact on cognitive outcomes and disease biomarkers. While preclinical studies strongly support curcumin's ability to reduce amyloid-β collection and improve synaptic plasticity, human trials have demonstrated mixed results. A key disadvantage is curcumin's low oral bioavailability, leading to insufficient central nervous system (CNS) exposure despite extreme doses. Some small-scale trials reported mild cognitive progress, but larger, placebo-controlled studies found no significant clinical benefit [35,4] Efforts to improve curcumin's bioavailability, such as nanoparticle formulations, liposomal carriers, and structural analogues, are ongoing and may lead to better clinical outcomes in future studies.

B. Ginkgo biloba in mental disorder

Extracts of Ginkgo biloba (EGb 761) represent one of the most widely tested phytochemicals in dementia-related conditions. Several randomised controlled trials have assessed its efficiency in AD, vascular dementia, and combined mental disorder. Evidence suggests ordinary cognitive improvements, particularly in attention, memory, and day-to-day living activities, when administered at therapeutic doses (120–240 mg/day) [3].

Few meta-analyses indicate that Ginkgo biloba can slow cognitive decline, while others highlight variability in study design, dosing, and patient heterogeneity as major confounders. Additionally, while mainly safe, potential interactions with anticoagulants require clinical caution [28,36] The irregularity in trial outcomes underscores the need for standardised dosing strategies and better patient stratification to clarify its clinical role.

C. Huperzine A in Alzheimer's Disease

Huperzine A, an alkaloid isolated from Huperzia serrata, acts as a reversible acetylcholinesterase inhibitor. Clinical trials in AD patients, specifically those conducted in China, have demonstrated positive effects on cognitive function, memory performance, and worldwide clinical status [28] Distinguished from conventional cholinesterase inhibitors, huperzine A appears well tolerated, with fewer gastrointestinal side effects. Basically, it crosses the blood–brain barrier effectively, contributing to its pharmacological relevance. However, most clinical studies have been relatively small and of short duration, limiting the strength of conclusions. Further big, multicentric trials with standardised protocols are required to establish its therapeutic utility and long-term security profile [28]

D. Resveratrol in Mild to Moderate Alzheimer's Disease

Resveratrol, a stilbene polyphenol found in grapes and berries, has been evaluated in human studies for its neuroprotective and antioxidant properties. A notable randomised, placebo-controlled trial in patients with mild-to-moderate AD assessed resveratrol supplementation and reported effects on cerebrospinal fluid (CSF) biomarkers. Particularly, resveratrol changed amyloid-β40 and matrix metalloproteinase-9 (MMP-9) levels, suggesting potential modulation of neuroinflammatory and amyloid pathways [6]. Still, regardless of biomarker changes, clinical improvements in cognition were limited, and issues with gastrointestinal side effects and dose growth remain unresolved. These findings highlight the translational gap between biomarker timbre and tangible clinical effects, emphasising the need for further refinement of treatment strategies.

E. Limitations of Existing Clinical Studies

Although phytochemicals show promise, various restraints restrict their integration into routine clinical practice. Firstly, many trials suffer from small sample sizes, short effect periods, and heterogeneous patient populations, lowering their statistical power. Secondly, variability in formulations, bioavailability, and dosing regimens complicates comparisons across studies [35,4,28,36,37]

IX. FUTURE PROSPECTS AND CHALLENGES

Phytochemicals offer a promising, multi-target approach to neurodegenerative disorders (NDs) by modulating oxidative stress, neuroinflammation, protein homeostasis, and mitochondrial function. However, extending the gap from experimental productiveness to reliable clinical therapies remains a bigger translational challenge. To harness the therapeutic potential of plant-derived bioactives, related advances are needed across clinical trial design, formulation and delivery, precision medicine, and combination therapeutic strategies. Below we explain these preferences and the practical obstacles that must be overcome, drawing on recent syntheses and empirical work [38,32,39,25,40,41]

A. Need for Large-Scale, Rigorous Clinical Trials

Current clinical evidence for many phytochemicals is restricted by small sample sizes, brief effects, various endpoints, and variable product formulations. To generate definitive efficacy and security data, randomised, multicentre, adequately powered trials with standardised effect measures are essential. Trials should include clinically meaningful endpoints (cognitive/functional scales, motor scores) plus biomarker substudies (imaging, CSF/plasma markers) to link biological activity with clinical effects. Embedding adaptive designs and stratified randomisation will help identify responsive subgroups while preserving effectiveness. Without such severe troubles, promising preclinical verdicts will stretch to produce uncertain clinical results and slow supervisory agreement [38,32]

B. Formulation and Delivery Strategies to Overcome Pharmacokinetic Barriers

A recurrent limitation of many phytochemicals (for example, curcumin, resveratrol, and miscellaneous polyphenols) is poor aqueous solubility, rapid metabolism, and restricted CNS availability. Advanced formulation technologies—containing nanoparticles, liposomes, solid lipid nanoparticles, polymeric micelles, and different nanocarriers—have shown in preclinical and early translational work that they can improve stability, prolong systemic circulation, and increase blood–brain barrier penetration [32,39]

These platforms also enable regulated release and targeted delivery, potentially reducing systemic exposure and reactions. Translational steps must address scale-up manufacturing, biocompatibility, reproducible quality control, and regulatory pathway clearness so that promising nanoformulations can progress through phase I–III testing [32,39,25]

C. Personalised Medicine and Biomarker-Guided Approaches

Neurodegenerative disorders are biologically heterogeneous; genetic background, comorbidity profiles, and molecular disease signatures influence response to therapies. Integrating pharmacogenomics, proteomic/metabolomic biomarkers, and clinical phenotyping can allow patient stratification and targeted application of phytochemical interventions. For example, patients with predominant neuroinflammatory signatures may, as a suggestion of choice, benefit from anti-inflammatory phytochemicals, whereas those with metabolic dysfunction may respond to mitochondria-focused compounds. Trials that prospectively layer participants and include biomarker endpoints will accelerate understanding of who benefits and why, enabling precision prevention and personalised adjunctive therapy [25,40]

D. Rational Combination Therapies

Phytochemicals often act on different cellular pathways, suggesting they may be most effective when used as adjuncts to standard treatments or in realistic combinations with additional bioactives. Synergistic combinations can amplify neuroprotective effects, allow dosage reductions of more toxic drugs, and target several disease mechanisms simultaneously. However, combination strategies require careful preclinical and clinical evaluation for pharmacokinetic and pharmacodynamic interplays, potential antagonism, and safety profiles. Adaptive and factorial clinical trial designs can efficiently evaluate mergers and recognise the most promising regimens for larger confirmatory studies [40,41]

E. Practical, Regulatory, and Manufacturing Challenges

Beyond scientific hurdles, reproducibility and standardisation remain critical problems. Natural product content varies with plant source, cultivation, harvest time, and extraction procedure; without standardised reference materials and GMP-grade manufacturing, inter-study comparisons remain difficult. Regulatory frameworks for complex natural-product formulations are less honest than for single-molecule drugs, complicating approval pathways. Intellectual property, funding incentives, and monetary interest in non-patentable natural products also influence translational momentum. Addressing these issues requires unity between academia, industry, and regulatory bodies to develop clear quality principles, validated analytical methods, and feasible commercial models [38,32,39,41]

X. CONCLUSION

Phytochemicals represent a promising multi-target strategy against neurodegenerative disorders by addressing the core pathological processes—oxidative stress, neuroinflammation, mitochondrial dysfunction, protein aggregation and synaptic failure. Preclinical evidence summarized in this review demonstrates that diverse classes (polyphenols, flavonoids, alkaloids, terpenoids) exert antioxidant, anti-amyloidogenic, anti-inflammatory and mitochondria-stabilizing effects and can modulate neurotransmission and proteostasis. However, clinical translation remains limited by poor bioavailability, variable formulations, short and underpowered trials, and inconsistent quality control of botanical materials. To move phytochemicals from nutraceutical promise to therapeutic reality, the field needs standardized botanical characterization, optimized delivery platforms (e.g., nanocarriers), biomarker-driven and adequately powered clinical trials, and rational combination or precision-medicine approaches. With these coordinated steps, phytochemicals could become effective adjuncts in multimodal management of neurodegenerative disease. And large-scale clinical trials to confirm therapeutic efficacy. With proper formulation and validation, phytochemicals could offer safe, effective, and affordable strategies for delaying the progression of neurodegenerative diseases and improving the quality of life of affected individuals

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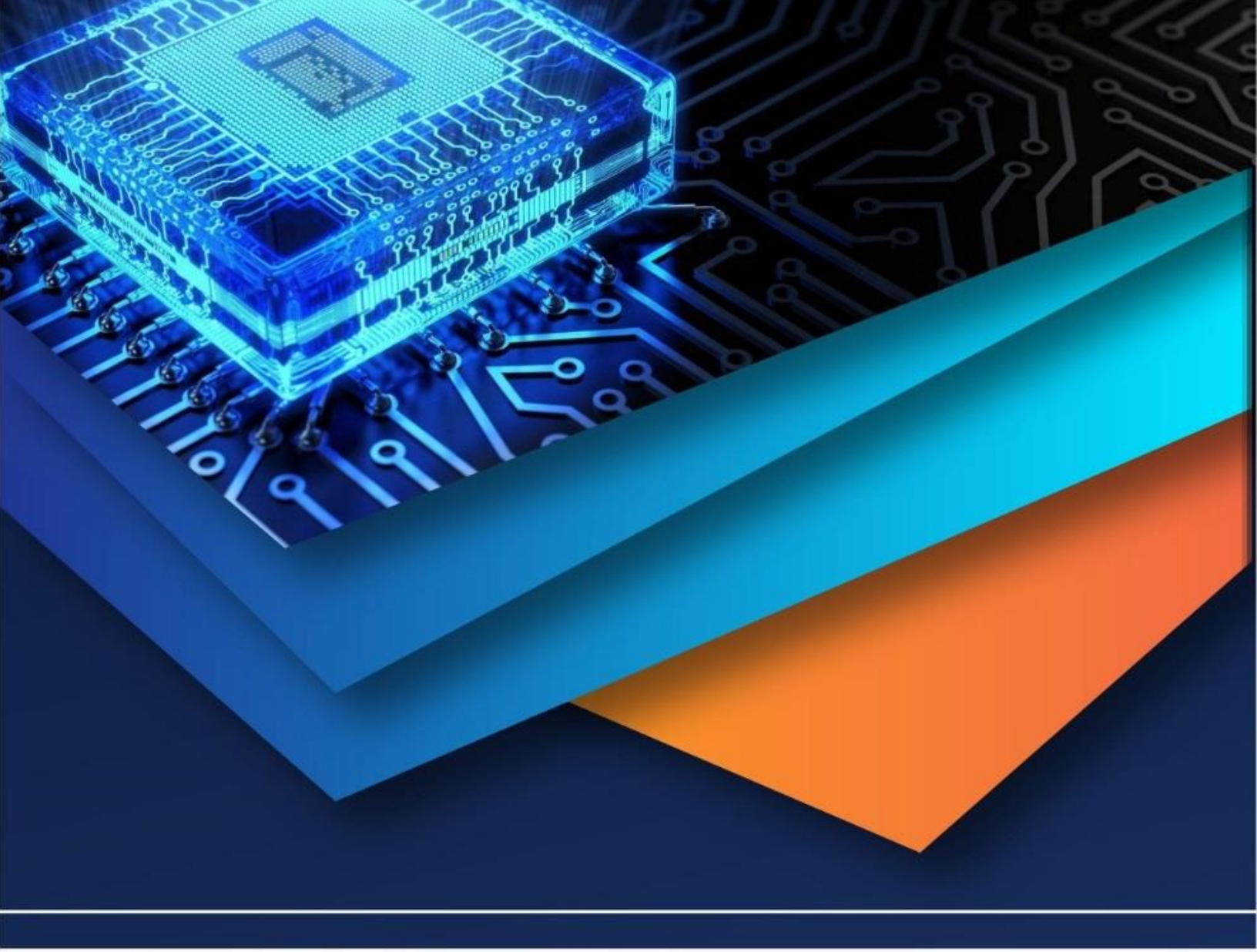

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