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Neuroprotective Effects of *Curcuma longa*: A Review of Its Role in Neurodegenerative Diseases

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Abstract

Curcuma longa (turmeric), a perennial herb notably utilized in traditional medication, has garnered big clinical attention due to its major bioactive compound, curcumin. Curcumin, well-known shows a couple of organic activities, together with antioxidant, anti-amyloidogenic, and neurodegenerative effects, making it a promising candidate for the management of neurodegenerative diseases (NDDs), which include Alzheimer's illness, Parkinson's disorder (PD), Huntington's sickness (HD), and amyotrophic lateral sclerosis (ALS). This evaluate consolidates current findings at the neuroprotective mechanisms of curcumin, its pharmacokinetics, bioavailability demanding situations, and its utility in medical settings. Emphasis is laid on in vitro and in vivo studies, as well as scientific trials assisting the efficacy of curcumin in mitigating neurodegenerative pathologies. Advances in nano formulation techniques aimed at enhancing its mind-targeted shipping are also mentioned. Universal, curcumin emerges as a multifaceted neuroprotective agent with translational relevance within the prevention and treatment of NDDs.

Keywords: *Curcuma longa*, curcumin, neuroprotection, neurodegenerative diseases, Alzheimer's disease, Parkinson's disease

INTRODUCTION

Neurodegenerative ailments (NDDs) constitute a hard and fast of progressive, disabling troubles characterized with the aid of the gradual degeneration of the structure and function of the valuable or peripheral nervous system [1]. Those sicknesses, collectively with Alzheimer's ailment (advert), Parkinson's sickness (PD), Huntington's ailment (HD), and amyotrophic lateral sclerosis (ALS), are important contributors to morbidity, disability, and mortality international. The pathogenesis of NDDs is multifactorial, regarding oxidative stress, mitochondrial dysfunction, neuroinflammation, protein aggregation, and excitotoxicity. Presently available healing options provide limited efficacy, targeting signs and symptoms in place of halting or reversing sickness progression. as a result, the search for safe, effective, and multifunctional neuroprotective retailers is a vital place of studies. In recent years, herbal products have gained significant interest as alternative or complementary strategies in the control of NDDs. Among them, *Curcuma longa* L. (circle of relatives: Zingiberaceae), normally referred to as turmeric, has emerged as a promising medicinal herb. extensively utilized in Indian and Southeast Asian traditional medicine systems, including Ayurveda and conventional Chinese language remedy (TCM), *Curcuma longa* long-standing reputation for treating a wide variety of ailments, together with inflammatory conditions, liver issues, and cognitive decline [2].

The principal active compound in *Curcuma longa* is curcumin, a hydrophobic polyphenol responsible for turmeric's characteristic yellow color. Curcumin has demonstrated extensive pharmacological activities, including antioxidant,

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anti-inflammatory, anti-amyloidogenic, antiapoptotic, and neurodegenerative properties. These biological effects position curcumin as a compelling candidate for combating the multifactorial pathology of NDDs [3].

PHYTOCHEMICAL PROFILE OF *CURCUMA LONGA*

Curcuma longa L., usually known as turmeric, is a rhizomatous herbaceous perennial plant significantly cultivated in tropical and subtropical regions. Its medicinal properties are primarily attributed to a diverse array of phytochemicals, particularly those concentrated in its underground rhizome. The major classes of bioactive compounds found in *Curcuma longa* include curcuminoids, volatile oils, and other secondary metabolites, each contributing distinct pharmacological activities, especially in the context of neuroprotection [4].

Curcuminoids

Curcuminoids are the most studied and pharmacologically massive constituents of *Curcuma longa*, accounting for about 2–8% of dry rhizome weight (Figure 1) [5]. The three essential curcuminoids are:

- *Curcumin (diferuloylmethane):* The predominant compound (~60–70% of total curcuminoids), well known for its strong antioxidant and anti-inflammatory properties [6].
- *Demethoxycurcumin:* A structural analogue of curcumin with potent anti-amyloid and anti-cancer activities.
- *Bisdemethoxycurcumin:* The least abundant but notable for its neuroprotective and anti-inflammatory effects [7].

These polyphenolic compounds exhibit extensive free radical scavenging abilities and can modulate multiple cellular signalling pathways involved in NDDs.

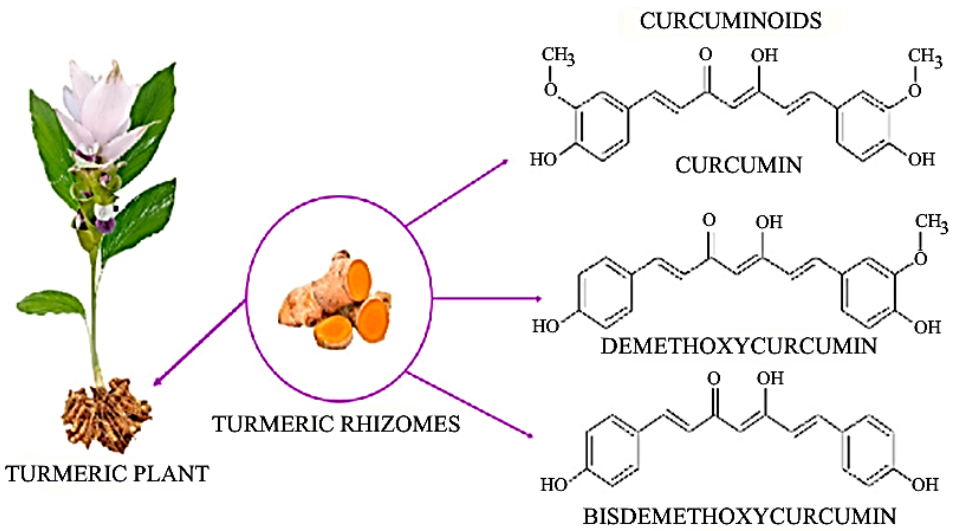

Figure 1. Curcuminoids are found in the rhizomes of turmeric.

Volatile Oils

Volatile critical oils make contributions to turmeric's features aroma and possess various healing properties, particularly anti-inflammatory and neurostimulatory effects. Key components include:

- Ar-turmerone.
- α-turmerone.
- Zingiberene.
- Atlantone.
- Curlone.

Among these, ar-turmerone has been shown to enhance neural stem cell proliferation and neurogenesis, further supporting the neuroprotective potential of turmeric [8].

Other Phytoconstituents

Apart from curcuminoids and volatile oils, *Curcuma longa* contains a range of additional constituents, such as:

- *Polysaccharides (e.g., ukonan):* Immunomodulatory and antioxidant effects.
- *Proteins and resins*.
- *Phenolic acids*, flavonoids, and sterols [9].

MECHANISMS OF NEUROPROTECTION

The neuroprotective efficacy of *Curcuma longa*, particularly its primary active compound curcumin, is attributed to a broad spectrum of mechanisms that counteract the pathological processes underlying NDDs. These mechanisms act synergistically to reduce neuronal loss, enhance synaptic function, and preserve cognitive and motor abilities. The multifactorial nature of neurodegeneration, which includes oxidative stress, neuroinflammation, protein aggregation, and mitochondrial disorder, makes curcumin an ideal candidate for intervention due to its pleiotropic biological effects (Figure 2) [10].

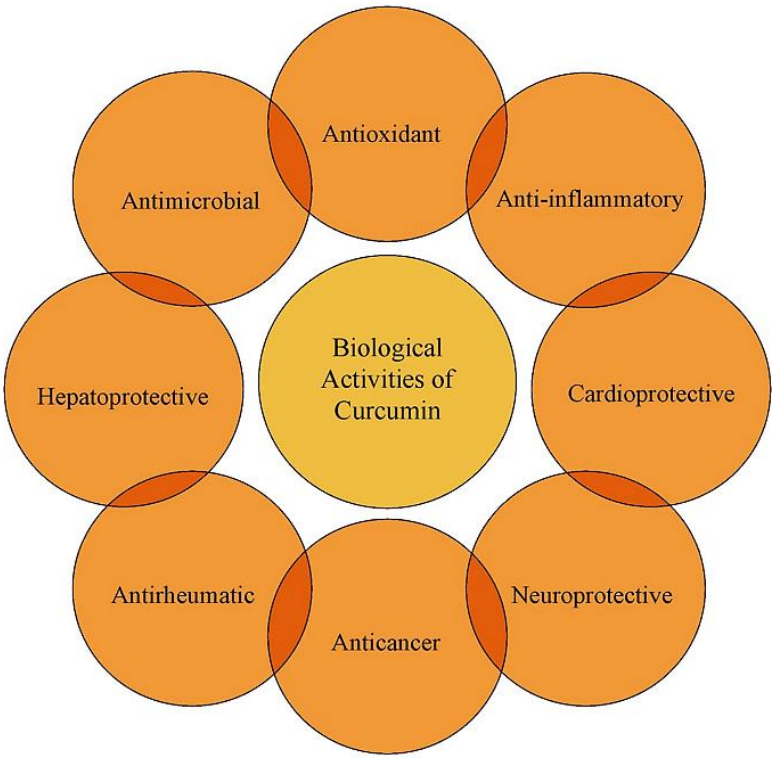

Figure 2. Schematic illustration of curcumin's biological activities.

Antioxidant Activity

One of the primary causes of neuronal harm in NDDs is oxidative stress due to the accumulation of reactive oxygen species (ROS) and reactive nitrogen species (RNS). Curcumin acts as a robust free radical scavenger by using the use of:

- Neutralizing ROS and RNS (e.g., superoxide, hydroxyl radicals).
- Enhancing endogenous antioxidant enzymes, such as:
  - Superoxide dismutase (SOD).
  - Catalase.
  - Glutathione peroxidase.

- Inhibiting lipid peroxidation and DNA damage [11].

Anti-Inflammatory Effects

Chronic neuroinflammation performs an essential function in the development of NDDs. Activated microglia and astrocytes release inflammatory cytokines (e.g., TNF-α, IL-1β, IL-6), which exacerbate neuronal death. Curcumin modulates inflammatory responses by means of:

- Inhibiting the activation of nuclear component-kappa B (NF-κB), a master regulator of contamination.
- Downregulating seasoned-inflammatory cytokines and enzymes, like COX-2 and iNOS.
- Suppressing microglial activation and infiltration.
- These actions contribute to a reduction in neuroinflammatory damage and preservation of neuronal homeostasis [12].

Anti-Amyloidogenic Activity

Protein aggregation, especially of amyloid-β (Aβ) and tau proteins in Alzheimer's disease (AD), is a hallmark of NDDs. Curcumin interferes with protein misfolding and aggregation through:

- Direct binding to Aβ plaques and tau fibrils, preventing aggregation.
- Promoting disaggregation of preformed Aβ plaques.
- Inhibiting β-secretase (BACE1) activity, thereby reducing Aβ production [13].

These mechanisms are critical for delaying or reversing amyloid-induced neurotoxicity and cognitive decline.

Mitochondrial Protection

Mitochondrial dysfunction leads to energy failure, ROS production, and apoptosis in neurodegenerative conditions. Curcumin protects mitochondria by:

- Stabilizing mitochondrial membrane potential (Δψm).
- Enhancing ATP production.
- Inhibiting mitochondrial permeability transition pore (mPTP) starts.
- Reducing cytochrome c release and apoptosis induction [14].

This supports cellular energy metabolism and prevents neuronal death.

Modulation of Apoptotic Pathways

Curcumin modulates the balance between seasoned-apoptotic and antiapoptotic factors.

- Downregulates pro-apoptotic proteins (Bax, caspases-3 and -9).
- Upregulates antiapoptotic proteins (Bcl-2).
- Prevents caspase-mediated neuronal apoptosis.

Enhancement of Neurogenesis and Synaptic Plasticity

Curcumin promotes brain restore and cognitive feature through:

- Upregulation of mind-derived neurotrophic thing and nerve increase element [15].
- Activation of cAMP response element-binding protein signalling.
- Enhancement of hippocampal neurogenesis and long-term potentiation. These effects support learning, memory formation, and structural brain plasticity.

CURCUMIN IN NEURODEGENERATIVE DISEASES

The multifactorial pathology of NDDs, including oxidative pressure, neuroinflammation, protein aggregation, and mitochondrial dysfunction, makes curcumin, with its huge-spectrum pharmacological houses, an attractive healing candidate. Numerous *in vitro*, *in vivo*, and emerging clinical studies have examined curcumin's efficacy in mitigating the pathophysiology and development of several primary NDDs, consisting of advert, PD, HD, and ALS [16].

Alzheimer's Disease (AD)

AD is the maximum normal shape of dementia, characterized via extracellular deposition of amyloid-beta (Aβ) plaques and intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein.

Curcumin's Effects in AD

- *Anti-amyloidogenic:* Binds to Aβ plaques, inhibits aggregation, and promotes disaggregation.
- *Anti-tau activity:* Inhibits tau hyperphosphorylation and stabilizes microtubules.
- *Anti-inflammatory:* Suppresses microglial activation and seasoned-inflammatory cytokine release.
- *Cognitive improvement:* Enhances memory and learning in transgenic mouse models (e.g., APP/PS1 mice).
- *Antioxidant:* Reduces ROS and lipid peroxidation in hippocampal neurons [17].

Parkinson's Disease (PD)

PD is marked thru the modern loss of dopaminergic neurons in the substantia nigra, main to motor disease and non-motor signs and signs.

Curcumin's Effects in PD

- *Neuroprotection:* Prevents dopaminergic neuronal death in MPTP and 6-OHDA models.
- *Antioxidant:* Reduces dopaminergic oxidative stress markers and restores glutathione levels.
- *Anti-inflammatory:* Inhibits microglial activation and cytokine release (e.g., TNF-α, IL-6).
- *Anti-aggregation:* Inhibits α-synuclein aggregation, a hallmark of Lewy body pathology [18].

Huntington's Disease (HD)

HD is a genetic NDD due to CAG repeat expansions in the HTT gene, main to mutant huntingtin (mHTT) protein aggregation.

Curcumin's Effects in HD

- *Anti-aggregatory:* Reduces mHTT protein aggregation.
- *Anti-excitotoxic:* Inhibits NMDA receptor overactivation and calcium influx.
- *Mitochondrial aid:* enhances mitochondrial function and ATP production.
- *Behavioral improvement:* Improves motor coordination and lifespan in R6/2 mice [19].

Amyotrophic Lateral Sclerosis (ALS)

ALS is a modern motor neuron disorder characterized with the aid of muscle atrophy, respiratory failure, and loss of life. The SOD1 gene mutation is commonly associated with familial ALS.

Curcumin's Effects in ALS

- *Antioxidant:* Protects against oxidative damage in spinal motor neurons.
- *Anti-inflammatory:* Decreases levels of TNF-α and IL-1β in ALS models.
- *Mitochondrial protection:* Preserves mitochondrial membrane integrity and ATP levels.
- *Neuroprotection:* Reduces motor neuron death and prolongs survival in SOD1-G93A transgenic mice [20].

PHARMACOKINETICS AND BIOAVAILABILITY CHALLENGES

Despite the promising neuroprotective effects demonstrated in preclinical models, the medical translation of curcumin stays restrained, basically due to its poor pharmacokinetic profile. Curcumin is characterized by way of low aqueous solubility, bad absorption from the gastrointestinal tract, rapid metabolism, and systemic elimination, all of which bring about extraordinarily low oral bioavailability. Following oral administration, curcumin undergoes widespread first-bypass metabolism within the liver and intestinal mucosa, where it's far conjugated into glucuronide and sulfate derivatives. Although these metabolites may retain some biological activity, their potency is generally inferior to that of the parent compound [21].

Furthermore, curcumin's poor stability in physiological pH and its limited ability to cross the blood-brain barrier (BBB) hinder its therapeutic effectiveness in central nervous system (CNS) disorders. These challenges necessitate the development of novel delivery systems to improve their systemic circulation, brain uptake, and therapeutic concentration at the target site.

Numerous strategies had been investigated to conquer those limitations. Co-administration with piperine, an alkaloid from black pepper, has been shown to enhance curcumin's bioavailability via 2000% with the aid of inhibiting glucuronidation. Further, nanotechnology-based strategies, inclusive of curcumin-loaded nanoparticles, liposomes, solid lipid nanoparticles, and polymeric micelles, have substantially improved curcumin's solubility, stability, and BBB permeability. Phospholipid complexes and curcumin conjugates with polyethylene glycol (PEG) have also demonstrated enhanced pharmacokinetics and therapeutic efficacy in experimental models of NDDs. These formulation advances hold great potential to unlock curcumin's clinical utility, especially in brain-targeted therapies [22].

CLINICAL STUDIES AND TRIALS

Curcumin's therapeutic potential in NDDs has prompted the initiation of several clinical investigations aimed at evaluating its efficacy and safety in human subjects. While preclinical studies consistently demonstrate its neuroprotective effects, translating these findings into clinical success has faced several hurdles, largely due to bioavailability issues and variability in study design [23].

Inside the context of Alzheimer's ailment (advert), early-phase clinical trials have yielded combined results. An outstanding randomized, placebo-controlled observe concerning individuals with slight-to-slight advert administered curcumin at doses of up to 4 g/day for 6 months. Although the treatment was well tolerated and showed modest biochemical effects, such as reduced plasma β-amyloid levels, no significant improvements in cognitive function were observed. However, in other observational studies, populations with high dietary turmeric intake (e.g., in India) have demonstrated lower incidences of cognitive decline, supporting the long-term neuroprotective role of curcumin as a dietary component. Newer trials using advanced formulations, like nanocurcumin or liposomal curcumin, have shown more promising results, with improved cognitive performance and biomarker modulation, although sample sizes remain small. In PD, clinical data are still in their infancy. Preliminary studies and case reports suggest curcumin's potential in reducing oxidative stress markers and improving non-motor signs, like mood and sleep disturbances. However, large-scale randomized controlled trials are lacking [24].

For general cognitive decline and age-associated memory impairment, several small-scale studies have demonstrated curcumin's ability to enhance attention, working memory, and mood in elderly participants. One such study using a highly bioavailable shape of curcumin (Theracurmin) mentioned large improvements in memory and reduced amyloid/tau accumulation as seen through PET imaging after 18 months of treatment. Despite these encouraging findings, the clinical landscape for curcumin remains underdeveloped. Most studies suffer from short durations, small sample sizes, lack of standardized curcumin formulations, and inconsistent endpoints. Moreover, the variability in bioavailability across different preparations complicates the interpretation of results [25].

NANO FORMULATIONS AND FUTURE DIRECTIONS

To overcome the inherent pharmacokinetic limitations of curcumin, including poor solubility, instability in physiological environments, rapid metabolism, and limited BBB penetration, researchers have turned to nanotechnology-primarily based transport structures. Nano formulations offer a promising technique to enhance the therapeutic efficacy of curcumin via enhancing its bioavailability, cellular uptake, and focused delivery to the brain (Table 1) [26].

Table 1. Nano formulations of curcumin and their relevance in neurodegenerative disease therapy [27].

Nanoformulation Type	Key Components	Advantages	Neuroprotective Relevance
Polymeric nanoparticles	PLGA, chitosan, PEG	Enhanced BBB permeability, sustained release	Improved cognitive and motor function in AD and PD animal models
Liposomal curcumin	Phospholipid bilayers	Improved systemic stability and bioavailability	Reduces neuroinflammation and Aβ burden in AD
Solid lipid nanoparticles	Lipid core (e.g., glyceryl monostearate)	High drug loading, good biocompatibility	Neuroprotection via reduced oxidative stress in PD
Curcumin-phytosomes	Curcumin + phospholipids	Enhanced absorption, better cellular uptake	Improved memory and reduced neurotoxicity in cognitive impairment models
Nanoemulsions	Oil-in-water or water-in-oil systems	High solubility, fast absorption	Potential use in fast-acting CNS-targeted delivery
Micelles	Amphiphilic block copolymers	Solubilization of hydrophobic curcumin	Delivers curcumin across BBB in Alzheimer's and ischemic brain models
Exosome-based systems	Exosome vesicles from cells	Biocompatible, immune-evasive, BBB-crossing	Emerging method for targeted brain delivery in NDDs
Dendrimers	Branched synthetic polymers	Controlled release, high drug loading	Investigated for curcumin co-delivery with neurotrophic factors

CONCLUSIONS

Curcuma longa, and its primary bioactive compound curcumin, has emerged as a compelling candidate inside the prevention and control of NDDs because of its pleiotropic pharmacological sports, consisting of antioxidant, anti-amyloidogenic, and neuroprotective consequences. Several preclinical research have shown curcumin's capability to counteract key pathological hallmarks associated with disorders, along with Alzheimer's illness, PD, HD, and ALS. Those findings underscore curcumin's capacity as a multitarget neuroprotective agent capable of modulating several interconnected mechanisms of neuronal injury.

But, no matter promising laboratory evidence, clinical translation remains limited through curcumin's poor pharmacokinetics and occasional systemic bioavailability. To address those demanding situations, a big selection of nano formulations has been advanced, enhancing curcumin's solubility, stability, and capability to move the BBB. Polymeric nanoparticles, liposomes, strong lipid nanoparticles, and phytosomes have proven unique promise in preclinical neurodegenerative fashions and are progressing closer to medical application.

Although early-phase clinical trials suggest that curcumin is safe and may offer modest cognitive benefits, further large-scale, well-controlled clinical studies are essential to confirm its efficacy and establish standardized dosing regimens. Future directions should focus on the integration of advanced nanotechnology with precision medicine strategies, ensuring brain-targeted delivery, and personalized treatment for neurodegenerative conditions.

In the end, curcumin holds considerable promise as an herbal therapeutic agent for neurodegeneration. With continued innovation in formulation science and rigorous clinical validation, it may soon become a valuable adjunct or even a frontline treatment option in the fight against debilitating NDDs.

REFERENCES

1. Gupta A, Sharma B. Neurodegenerative diseases (ND): An introduction. In: Synaptic Plasticity in Neurodegenerative Disorders. CRC Press; 2025. pp. 3–20.

2. Kaliyadasa E, Samarasinghe BA. A review on golden species of Zingiberaceae family around the world: Genus Curcuma. Afr J Agric Res. 2019;14(9):519–531. doi: 10.5897/AJAR2018.13755.
3. Vo TS, Vo TT, Vo TT, Lai TN. Turmeric (*Curcuma longa* L.): Chemical components and their effective clinical applications. J Turk Chem Soc A Chem. 2021;8(3):883–898. doi: 10.18596/jotcsa.913136.
4. Chanda S, Ramachandra TV. Phytochemical and pharmacological importance of turmeric (*Curcuma longa*): A review. Res Rev J Pharmacol. 2019;9(1):16–23.
5. Fuloria S, Mehta J, Chandel A, Sekar M, Rani NN, Begum MY, et al. A comprehensive review on the therapeutic potential of Curcuma longa Linn. in relation to its major active constituent curcumin. Front Pharmacol. 2022;13:820806. doi: 10.3389/fphar.2022.820806.
6. Helson L. Curcumin (diferuloylmethane) delivery methods: A review. Biofactors. 2013;39(1):21–26. doi: 10.1002/biof.1080.
7. Jayaprakasha GK, Rao LJ, Sakariah KK. Antioxidant activities of curcumin, demethoxycurcumin and bisdemethoxycurcumin. J Agric Food Chem. 2002;50(13):3668–3672. doi: 10.1021/jf025506a.
8. Orellana-Paucar AM. Turmeric essential oil constituents as potential drug candidates: A comprehensive overview of their individual bioactivities. Molecules. 2024;29(17):4210. doi: 10.3390/molecules29174210.
9. Hooda P, Malik R, Bhatia S, Al-Harrasi A, Najmi A, Zoghebi K, et al. Phytoimmunomodulators: A review of natural modulators for complex immune system. Heliyon. 2024;10(1):e23790. doi: 10.1016/j.heliyon.2023.e23790.
10. Nebrisi EE. Neuroprotective activities of curcumin in Parkinson's disease: A review of the literature. Int J Mol Sci. 2021;22(20):11248. doi: 10.3390/ijms222011248.
11. Islam MT. Oxidative stress and mitochondrial dysfunction-linked neurodegenerative disorders. Neurol Res. 2017;39(1):73–82. doi: 10.1080/01616412.2016.1251711.
12. Rauf A, Badoni H, Abu-Izneid T, Olatunde A, Rahman MM, Painuli S, et al. Neuroinflammatory markers: Key indicators in the pathology of neurodegenerative diseases. Molecules. 2022;27(10):3194. doi: 10.3390/molecules27103194.
13. Candelise N, Scaricamazza S, Salvatori I, Ferri A, Valle C, Manganelli V, et al. Protein aggregation landscape in neurodegenerative diseases: Clinical relevance and future applications. Int J Mol Sci. 2021;22(11):6016. doi: 10.3390/ijms22116016.
14. Bhat AH, Dar KB, Anees S, Zargar MA, Masood A, Sofi MA, et al. Oxidative stress, mitochondrial dysfunction and neurodegenerative diseases; a mechanistic insight. Biomed Pharmacother. 2015;74:101–110. doi: 10.1016/j.biopha.2015.07.025.
15. Namgyal D, Ali S, Mehta R, Sarwat M. The neuroprotective effect of curcumin against Cd-induced neurotoxicity and hippocampal neurogenesis promotion through CREB-BDNF signaling pathway. Toxicology. 2020;442:152542. doi: 10.1016/j.tox.2020.152542.
16. Bagheri H, Ghasemi F, Barreto GE, Rafiee R, Sathyapalan T, Sahebkar A. Effects of curcumin on mitochondria in neurodegenerative diseases. Biofactors. 2020;46(1):5–20. doi: 10.1002/biof.1566.
17. Sajjad R, Arif R, Shah AA, Manzoor I, Mustafa G. Pathogenesis of Alzheimer's disease: Role of amyloid-beta and hyperphosphorylated tau protein. Indian J Pharm Sci. 2018;80(4):581–591. doi: 10.4172/pharmaceutical-sciences.1000397.
18. Chaudhuri KR, Schapira AH. Non-motor symptoms of Parkinson's disease: Dopaminergic pathophysiology and treatment. Lancet Neurol. 2009;8(5):464–474. doi: 10.1016/S1474-4422(09)70068-7.
19. Podvin S, Reardon HT, Yin K, Mosier C, Hook V. Multiple clinical features of Huntington's disease correlate with mutant HTT gene CAG repeat lengths and neurodegeneration. J Neurol. 2019;266:551–564. doi: 10.1007/s00415-018-8940-6.
20. Chico L, Ienco EC, Bisordi C, Lo Gerfo A, Petrozzi L, Petrucci A, et al. Amyotrophic lateral sclerosis and oxidative stress: A double-blind therapeutic trial after curcumin supplementation. CNS Neurol Disord Drug Targets. 2018;17(10):767–779. doi: 10.2174/1871527317666180720162029.

21. Liu W, Zhai Y, Heng X, Che FY, Chen W, Sun D, et al. Oral bioavailability of curcumin: Problems and advancements. J Drug Target. 2016;24(8):694–702. doi: 10.3109/1061186X.2016.1157883.
22. Lee SH, Kim HY, Back SY, Han HK. Piperine-mediated drug interactions and formulation strategy for piperine: Recent advances and future perspectives. Expert Opin Drug Metab Toxicol. 2018;14(1):43–57. doi: 10.1080/17425255.2018.1418854.
23. Sharma P, Mishra R, Sharma B, Kumari A, Modgil S. Health sciences: Advancing healthcare through interdisciplinary approaches and innovation. Int J Multidiscip Appl Bus Educ Res. 2025;6(5):2309–2316. doi: 10.11594/ijmaber.06.05.17.
24. Mishra R, Verma A, Sharma B, Sharma P. An overview of transdermal patches: Opportunities and obstacles in modern drug delivery. Asian J Res Med Pharm Sci. 2025;14(3):18–29. doi: 10.9734/ajrimps/2025/v14i3314.
25. World J. Pharm. Res. 2015; 14(08):618-631.
26. Harry A. Revolutionizing healthcare: the transformative role of artificial intelligence in the health sector. BULLET: Jurnal Multidisiplin Ilmu. 2023 May;2(2):326-35.
27. Yadav A, Sharma S, Wani SN, Kaushal K, Sharma P. Development of oral herbal male contraceptive. Curr Asp Pharm Res Dev. 2022;8:77–88. doi: 10.9734/bpi/caprd/v8/3416E.

