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NATURAL BIOACTIVE COMPOUNDS AS MULTI-TARGET MODULATORS OF NEUROINFLAMMATION AND NEURODEGENERATION: A SHORT REVIEW

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ABSTRACT

Neurodegenerative disorders are multifactorial conditions characterized by progressive neuronal loss, cognitive decline and functional impairment, driven by interconnected processes such as neuroinflammation, oxidative stress, protein aggregation, impaired autophagy and DNA damage. Owing to the limited success of single-target therapies, increasing attention has been directed toward natural bioactive compounds with multi-target therapeutic potential. This short review highlights the neuroprotective significance of Bacopa monnieri, curcumin, quercetin and asiatic acid, with emphasis on their modulatory effects on key molecular pathways involved in neuroinflammation and neurodegeneration. Available evidence indicates that these compounds influence important targets including NLRP3, Nrf2, GSK-3β, HDAC6, PARP1 and mTOR through both direct and indirect mechanisms. Their actions are associated with suppression of inflammatory signaling, attenuation of oxidative stress, reduction of protein aggregation, regulation of autophagy and enhancement of neuronal survival. Among these compounds, curcumin and quercetin appear to have relatively broader mechanistic evidence, whereas Bacopa monnieri and asiatic acid show promising neuroprotective effects mainly through upstream signaling modulation and antioxidant activity. Despite these encouraging findings, challenges such as poor bioavailability, limited structural validation and insufficient clinical evidence continue to hinder their translational application. Overall, these natural compounds represent promising multi-target agents for the management of neurodegenerative disorders, although further mechanistic and clinical studies are required to establish their therapeutic utility.

KEYWORDS: Neurodegeneration; Neuroinflammation; Natural bioactive compounds; Bacopa monnieri; Curcumin; Quercetin; Asiatic acid; NLRP3; Nrf2; GSK-3β; HDAC6; PARP1; mTOR

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1. INTRODUCTION

Neurodegenerative disorders are complex and progressive conditions characterized by neuronal loss, cognitive decline and functional impairment. Their development is driven by multiple interconnected mechanisms, including chronic neuroinflammation, oxidative stress, mitochondrial dysfunction, abnormal protein aggregation, impaired autophagy and DNA damage. Because these pathological events interact closely, therapeutic approaches based on a single molecular target often provide limited benefit (1-2). In recent years, natural bioactive compounds have received increasing attention as potential neuroprotective agents due to their broad pharmacological actions, relatively low toxicity and ability to regulate several disease-related pathways simultaneously. Among these compounds, *Bacopa monnieri*, curcumin, quercetin and asiatic acid have emerged as promising candidates (3). The literature provided indicates that these agents can influence major molecular targets associated with neuroinflammation and neurodegeneration, including NLRP3, Nrf2, GSK-3β, HDAC6, PARP1 and mTOR. Their effects are associated with suppression of inflammatory signaling, enhancement of antioxidant defense, reduction of protein aggregation, modulation of autophagy and preservation of neuronal survival (4-7). These properties suggest that such compounds may offer a multi-target therapeutic strategy for diseases in which oxidative stress, inflammation and neuronal dysfunction coexist. This short review summarizes the mechanistic relevance and therapeutic significance of selected natural bioactive compounds in the regulation of key pathways involved in neuroinflammation and neurodegeneration (8). It further highlights their comparative potential, major limitations and future prospects for translational application in the management of neurodegenerative disorders. Natural bioactive compounds such as *Bacopa monnieri*, curcumin, quercetin and asiatic acid show promising relevance as multi-target neuroprotective

agents in neuroinflammation and neurodegeneration. This graphical abstract highlights how these compounds act on interconnected molecular targets, including NLRP3, Nrf2, GSK-3β, HDAC6, PARP1 and mTOR, to regulate major pathological processes such as inflammation, oxidative stress, protein aggregation, impaired autophagy and DNA damage (9-11). By modulating these pathways, the selected phytochemicals may reduce neuronal injury, improve cellular homeostasis and support neuronal survival (Figure 1). The figure summarizes the central concept of this review: unlike single-target therapies, natural compounds may offer broader therapeutic value by simultaneously influencing multiple disease-driving mechanisms involved in progressive neurodegenerative disorders.

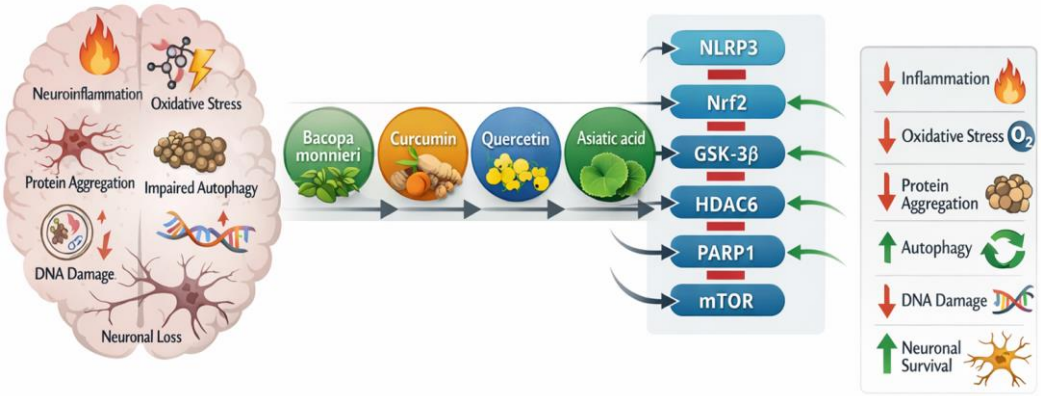

Figure 1. Graphical Abstract showing Natural Bioactive Compounds as Multi-Target Modulators of Neuroinflammation and Neurodegeneration

2. MODULATION OF INFLAMMATION AND OXIDATIVE STRESS

Inflammation and oxidative stress are central contributors to the onset and progression of neurodegenerative disorders. Persistent activation of inflammatory pathways promotes neuronal injury, while excessive production of reactive oxygen species damages lipids, proteins and nucleic acids, further accelerating neurodegeneration (12). In this context, the NLRP3 inflammasome and the Nrf2 signaling pathway represent two major and interrelated molecular systems involved in inflammatory and oxidative stress responses. NLRP3 promotes the maturation of pro-inflammatory cytokines such as interleukin-1β and interleukin-18, whereas Nrf2 functions as a master regulator of antioxidant and cytoprotective gene expression. Dysregulation of these pathways is strongly linked with chronic neuroinflammation and redox imbalance (13). The reviewed compounds demonstrate notable potential in modulating both pathways. *Bacopa monnieri* appears to suppress NLRP3 activation mainly through indirect mechanisms, including reduction of mitochondrial dysfunction, attenuation of reactive oxygen species generation and enhancement of mitophagy. It also promotes antioxidant defence by activating Nrf2-related signaling and increasing the expression of protective enzymes. Curcumin shows broader mechanistic relevance, as it is reported to inhibit NLRP3 activation while also activating Nrf2 through disruption of the NRF2–KEAP1 complex (14-17). Quercetin similarly exhibits dual activity by reducing inflammatory signalling and enhancing antioxidant responses, partly through interaction with Keap1 and suppression of oxidative stress. Asiatic acid also contributes to the regulation of these pathways, mainly by decreasing mitochondrial oxidative damage, modulating NF-κB-related inflammatory signaling and promoting Nrf2-mediated cytoprotection. Taken together, these findings indicate that the anti-inflammatory and antioxidant effects of these natural compounds are among their most significant therapeutic attributes (18-20). Their ability to simultaneously suppress inflammasome-driven inflammation and strengthen endogenous antioxidant defenses suggests an important role in limiting neuronal injury. This dual action is especially valuable in neurodegenerative disorders, where inflammation and oxidative stress continuously reinforce one another and contribute to disease progression.

3. REGULATION OF PROTEINOPATHY AND NEURONAL SIGNALING

One of the key problems in neurodegenerative diseases, especially Alzheimer's disease, is the buildup of misfolded proteins, abnormal chemical modifications and disrupted signals inside nerve cells (21). Two important molecules, GSK-3β and HDAC6, play central roles in these harmful processes (22). GSK-3β is known to drive excessive changes in the tau protein, which leads to the formation of tangles in brain cells, triggers the death of neurons and disrupts communication between them. Meanwhile, HDAC6 helps control the structure and function of the cell's scaffolding system, manages the removal

of damaged proteins and supports the cell's natural cleanup mechanisms (23). When these two molecules are out of balance, brain cells become more unstable and the buildup of damaged proteins accelerates, making the disease worse (24). Recent research suggests that natural compounds may help restore this balance. *Bacopa monnieri*, for instance, appears to regulate GSK-3β activity, possibly by activating protective signalling routes in the cell. This action can help reduce tau-related damage and support nerve cell survival (25). It may also positively affect HDAC6, leading to stronger cell structures and better removal of misfolded proteins (26). Curcumin has shown promise for acting directly on the active site of GSK-3β and may also block HDAC6's harmful activity (27). These effects could help prevent abnormal tau buildup, improve the movement of vital materials along nerve fibres and reduce protein clumping. Quercetin seems to work in a similar way, helping to stabilize important cellular pathways and support the effective handling of damaged proteins (28). Although the evidence is still emerging, asiatic acid may also play a helpful role by supporting healthy signalling inside neurons, maintaining the cell's antioxidant defences and encouraging the clearance of unwanted proteins (29). Taken together, these findings underscore the potential of natural bioactive compounds to tackle the underlying problems that cause nerve cell damage and protein buildup in the brain (30). By influencing both GSK-3β and HDAC6, these compounds offer protection that goes beyond just fighting inflammation or oxidative stress. Instead, they may help keep the brain's internal structure stable, prevent the buildup of harmful proteins and ultimately slow the worsening of neurodegenerative diseases.

4. CONTROL OF AUTOPHAGY, DNA DAMAGE AND CELLULAR SURVIVAL

The progression of neurodegenerative diseases is also strongly linked to problems with the cell's own cleanup system, increased DNA damage and loss of energy balance within neurons (31). Two molecules, PARP1 and mTOR, play crucial roles in these processes. PARP1 helps cells sense and repair DNA damage, but when it is overactive, it can drain the cell's energy stores, ultimately leading to cell death. On the other hand, mTOR acts as a master controller of cell growth, metabolism and the cell's natural recycling process known as autophagy. If mTOR becomes too active, it can block this cleanup process, causing harmful proteins and damaged cell parts to build up (32). When these pathways are out of balance, nerve cells become more vulnerable and the disease can progress more rapidly (33). Recent findings suggest that natural compounds may help restore balance to these systems. *Bacopa monnieri*, for example, seems to support healthy PARP1 and mTOR function by reducing oxidative DNA damage, helping cells conserve energy and activating protective signalling routes inside the cell (34). These effects may lower the risk of energy depletion and encourage the removal of damaged proteins. Curcumin appears to go even further, with research indicating that it can directly block PARP1's activity and may also interact with mTOR to help reactivate the cell's cleanup machinery (35). Quercetin shows similar benefits, as it can reduce DNA damage, limit the harmful effects of PARP1 overactivity and support proper autophagy by influencing mTOR pathways (36). Asiatic acid may also help nerve cells survive by fighting oxidative stress, dampening harmful signalling and helping regulate the PI3K/Akt/mTOR system, although more research is needed to fully understand these effects (37). Ultimately, these observations suggest that natural bioactive compounds may offer protection to brain cells in multiple ways. They not only help reduce inflammation and oxidative stress but also maintain DNA stability, support the removal of damaged materials and keep the cell's energy system running smoothly. This kind of broad, multi-level protection is especially important in neurodegenerative diseases, where nerve cell survival depends on both keeping the internal environment clean and resisting stress-induced damage. The impact these compounds have on PARP1 and mTOR adds meaningful value to their overall potential as neuroprotective agents (38).

5. COMPARATIVE THERAPEUTIC SIGNIFICANCE OF SELECTED COMPOUNDS

Among the compounds discussed, curcumin and quercetin appear to have relatively broader mechanistic support across multiple targets involved in neuroinflammation and neurodegeneration (39). Both compounds are repeatedly associated with modulation of inflammatory signalling, antioxidant defence, kinase regulation, proteostasis, autophagy and cell survival pathways (40). In particular, they show stronger evidence for possible direct interaction with targets such as NLRP3, GSK-3β, HDAC6, PARP1 and mTOR (41), while also exerting indirect protective effects through suppression of reactive oxygen species, inhibition of NF-κB signaling and activation of Nrf2-related antioxidant responses (42). This broad spectrum of activity makes them especially attractive as multi-target neuroprotective agents.

Bacopa monnieri and asiatic acid also demonstrate important therapeutic potential, although their evidence base is somewhat more dependent on indirect pathway modulation than on well-defined structural interaction studies (43). *Bacopa monnieri* is particularly notable for its antioxidant, anti-inflammatory, cognitive-enhancing and mitochondrial protective effects, which support its relevance in neurodegenerative conditions (44). However, variability in phytochemical composition and standardization remains an important limitation (45). Asiatic acid similarly exhibits promising neuroprotective properties through regulation of oxidative stress, inflammatory signaling and autophagy-related pathways, but direct molecular validation is comparatively less developed (46). Even so, both compounds remain significant because they contribute to upstream regulation of disease-driving mechanisms and may complement better-characterized phytochemicals.

Ultimately, the comparative significance of these compounds lies in their shared ability to influence multiple interconnected targets rather than a single isolated pathway (47). Curcumin and quercetin may currently appear more mechanistically mature, whereas *Bacopa monnieri* and asiatic acid offer strong supportive value through broader neuroprotective and antioxidant actions. This comparative profile suggests that natural compounds with multi-target effects may provide a more realistic therapeutic strategy for complex disorders such as neurodegeneration, where inflammation, oxidative stress, proteinopathy and impaired cellular survival are closely intertwined (48).

6. CURRENT LIMITATIONS AND FUTURE DIRECTIONS

Despite the promising therapeutic relevance of these natural bioactive compounds, several important limitations continue to restrict their translational application. One of the most consistent challenges is poor bioavailability, particularly for curcumin and quercetin, which undergo rapid metabolism and show limited systemic stability. Similar concerns also apply to asiatic acid because of its poor solubility, while *Bacopa monnieri* presents additional challenges related to variability in phytochemical composition and lack of standardized formulations. Another major limitation is the insufficient availability of direct molecular evidence for several compound–target interactions. Although many studies suggest favorable mechanistic effects, detailed structural validation through advanced docking, molecular dynamics simulations and high-resolution binding studies remains inadequate for several pathways. In addition, much of the currently available evidence is derived from in silico analyses, cell-based experiments and animal models, while robust clinical validation remains limited. This gap makes it difficult to determine the true therapeutic efficacy, safety, optimal dosage and long-term applicability of these compounds in human neurodegenerative disorders. Future research should therefore focus on standardizing compound formulations, improving pharmacokinetic properties through nanoformulation and targeted delivery systems and conducting detailed mechanistic investigations to confirm direct target engagement. Well-designed in vivo studies and clinical trials are also necessary to evaluate translational potential and establish these natural compounds as credible therapeutic candidates.

7. CONCLUSION

Natural bioactive compounds such as *Bacopa monnieri*, curcumin, quercetin and asiatic acid show considerable promise as multi-target modulators of neuroinflammation and neurodegeneration. The available literature indicates that these compounds influence several interconnected molecular pathways, including NLRP3, Nrf2, GSK-3β, HDAC6, PARP1 and mTOR, which are closely involved in inflammation, oxidative stress, protein aggregation, autophagy, DNA damage and neuronal survival. Their ability to act across multiple pathological mechanisms makes them particularly relevant for complex neurodegenerative disorders, where single-target therapeutic strategies often remain insufficient. Among the reviewed compounds, curcumin and quercetin appear to possess relatively broader mechanistic evidence, while *Bacopa monnieri* and asiatic acid contribute significant neuroprotective value through antioxidant, anti-inflammatory and upstream regulatory effects. Despite these encouraging findings, limitations related to poor bioavailability, insufficient structural validation and limited clinical evidence continue to hinder their therapeutic translation. Finally, these natural compounds represent promising candidates for future neuroprotective interventions, but further mechanistic studies and well-designed clinical investigations are required to confirm their efficacy and support their application in disease management.

REFERENCES

1. Soto, C., & Pritzkow, S. (2018). Protein misfolding, aggregation and conformational strains in neurodegenerative diseases. Nature Neuroscience, 21(10), 1332-1340. https://doi.org/10.1038/s41593-018-0235-9
2. Heneka, M. T., Golenbock, D. T., & Latz, E. (2015). Innate immunity in Alzheimer's disease. Nature Immunology, 16(3), 229-236. https://doi.org/10.1038/ni.3102
3. Cummings, J. L., Morstorf, T., & Zhong, K. (2014). Alzheimer's disease drug-development pipeline: Few candidates, frequent failures. Alzheimer's Research & Therapy, 6(4), 37. https://doi.org/10.1186/alzrt269
4. Howes, M. J. R., & Perry, E. (2011). The role of phytochemicals in the treatment and prevention of dementia. Drugs & Aging, 28(6), 439-468. https://doi.org/10.2165/11591310-000000000-00000
5. Singh, H. K., & Dhawan, B. N. (1997). Neuropsychopharmacological effects of the Ayurvedic nootropic Bacopa monniera Linn. (Brahmi). Indian Journal of Pharmacology, 29(5), S359-S365.
6. Choi, S. H., & Bosetti, F. (2009). Cyclooxygenase-1 null mice show reduced neuroinflammation in response to beta-amyloid. Aging, 1(2), 234-244. https://doi.org/10.18632/aging.100020
7. Ullah, R., Ali, T., Ullah, N., Kim, M. O., & Caffeine Prevents Oxidative Stress and Neuroinflammation by Regulating Nrf2/TLR4/NF-κB Signaling Pathway in a Mouse Model of Parkinson's Disease. Aging and Disease, 11(6), 1273-1289. https://doi.org/10.14336/AD.2019.1129
8. Butterfield, D. A., & Halliwell, B. (2019). Oxidative stress, dysfunctional glucose metabolism and Alzheimer disease. Nature Reviews Neuroscience, 20(3), 148-160. https://doi.org/10.1038/s41583-019-0132-6
9. Ramesh, G., & Biju, T. (2020). Phytochemicals as multi-target therapeutic agents for neurodegenerative diseases. Current Neuropharmacology, 18(7), 636-655. https://doi.org/10.2174/1570159X18666200128114223
10. Heneka, M. T., McManus, R. M., & Latz, E. (2018). Inflammasome signalling in brain function and neurodegenerative disease. Nature Reviews Neuroscience, 19(10), 610–621. https://doi.org/10.1038/s41583-018-0055-7
11. Butterfield, D. A., & Halliwell, B. (2019). Oxidative stress, dysfunctional glucose metabolism and Alzheimer disease. Nature Reviews Neuroscience, 20(3), 148–160. https://doi.org/10.1038/s41583-019-0132-6
12. Swanson, K. V., Deng, M., & Ting, J. P.-Y. (2019). The NLRP3 inflammasome: molecular activation and regulation to therapeutics. Nature Reviews Immunology, 19(8), 477–489. https://doi.org/10.1038/s41577-019-0165-0
13. Yamamoto, M., Kensler, T. W., & Motohashi, H. (2018). The Keap1–Nrf2 system: a thiol-based sensor–effector apparatus for maintaining redox homeostasis. Physiological Reviews, 98(3), 1169–1203. https://doi.org/10.1152/physrev.00023.2017
14. Ahmed, S. M. U., Luo, L., Namani, A., Wang, X. J., & Tang, X. (2017). Nrf2 signaling pathway: Pivotal roles in inflammation. Biochimica et Biophysica Acta (BBA) - Molecular Basis of Disease, 1863(2), 585–597. https://doi.org/10.1016/j.bbadis.2016.11.005
15. Limpeanchob, N., Jaipan, S., Rattanakaruna, S., Phrompittayarat, W., & Ingkaninan, K. (2008). Neuroprotective effect of Bacopa monnieri on beta-amyloid-induced cell death in primary cortical culture. Journal of Ethnopharmacology, 120(1), 112–117. https://doi.org/10.1016/j.jep.2008.07.046
16. Uabundit, N., Wattanathorn, J., Mucimapura, S., & Ingkaninan, K. (2010). Cognitive enhancement and neuroprotective effects of Bacopa monnieri in Alzheimer's disease model. Journal of Ethnopharmacology, 127(1), 26–31. https://doi.org/10.1016/j.jep.2009.09.056
17. Zhang, S., Liu, X., Bian, H., Zhang, Y., & Sun, L. (2021). Curcumin ameliorates neuroinflammation by suppressing NLRP3 inflammasome activation and promoting microglial M2 polarization via the NRF2 pathway. Journal of Neuroinflammation, 18(1), Article 26. https://doi.org/10.1186/s12974-021-02065-w
18. Wang, D., Liu, L., Zhu, X., Wu, W., Wang, Y., & Wang, X. (2020). Quercetin suppresses NLRP3 and Nrf2 pathways in microglia to attenuate neuroinflammation. International Immunopharmacology, 84, 106511. https://doi.org/10.1016/j.intimp.2020.106511

19. Chen, L., Hu, L., Chan, T. H., & Hu, Y. (2012). Asiatic acid protects primary neurons against mitochondrial toxicity via antioxidant pathway. Neurochemistry International, 61(3), 360–369. https://doi.org/10.1016/j.neuint.2012.05.008
20. Ramesh, G., & Biju, T. (2020). Phytochemicals as multi-target therapeutic agents for neurodegenerative diseases. Current Neuropharmacology, 18(7), 636–655. https://doi.org/10.2174/1570159X18666200128114223
21. Wu, A. G., Wang, X. Y., Zhou, X. D., Qiao, G., Jiang, T., & Chen, S. Y. (2019). Neuroprotective effects of curcumin in a 6-hydroxydopamine-induced Parkinson's disease model via the Wnt/β-catenin signaling pathway. CNS Neuroscience & Therapeutics, 25(4), 367–376. https://doi.org/10.1111/cns.13060Opens a new window
22. Nabavi, S. F., Sureda, A., Daglia, M., Nabavi, S. M., & Moghaddam, A. H. (2015). Neuroprotective effects of quercetin as an anti-Alzheimer agent: From chemistry to molecular targets. Current Topics in Medicinal Chemistry, 15(23), 2149–2155. https://doi.org/10.2174/1568026615666150722102755Opens a new window
23. Zhang, Y., Gong, X., Lu, X., & Xu, J. (2021). Asiatic acid protects against cognitive and neuronal impairments induced by D-galactose in mice via regulating the Nrf2/HO-1 signaling pathway. International Immunopharmacology, 90, 107181. https://doi.org/10.1016/j.intimp.2020.107181Opens a new window
24. Zeng, K. W., Fu, H., Liu, G. X., Wang, X. M., & Tu, P. F. (2011). Asiatic acid inhibits neuroinflammation and attenuates microglia activation. Neurochemistry International, 58(3), 403–410. https://doi.org/10.1016/j.neuint.2010.12.005Opens a new window
25. Dey, A., & De, J. (2015). Neuroprotective therapeutics from botanicals and phytochemicals against Huntington's disease and Alzheimer's disease. CNS & Neurological Disorders - Drug Targets, 14(8), 1011–1027. https://doi.org/10.2174/1871527314666150722100955Opens a new window
26. Bhat, A. H., Dar, K. B., Anees, S., Zargar, M. A., Masood, A., Sofi, M. A., & Ganie, S. A. (2015). Oxidative stress, mitochondrial dysfunction and neurodegenerative diseases; a mechanistic insight. Biomedicine & Pharmacotherapy, 74, 101–110. https://doi.org/10.1016/j.biopha.2015.07.025Opens a new window
27. Pohanka, M. (2018). Oxidative stress in Alzheimer disease as a target for therapy. Bratislava Medical Journal, 119(9), 543–553. https://doi.org/10.4149/BLL_2018_097Opens a new window
28. Morris, M. C., Evans, D. A., Tangney, C. C., Bienias, J. L., & Wilson, R. S. (2006). Associations of vegetable and fruit consumption with age-related cognitive change. Neurology, 67(8), 1370–1376. https://doi.org/10.1212/01.wnl.0000240224.38978.faOpens a new window
29. Vauzour, D., Vafeiadou, K., Rodriguez-Mateos, A., Rendeiro, C., & Spencer, J. P. (2008). The neuroprotective potential of flavonoids: A multiplicity of effects. Genes & Nutrition, 3(3-4), 115–126. https://doi.org/10.1007/s12263-008-0091-4Opens a new window
30. Anand, P., Kunnumakkara, A. B., Newman, R. A., & Aggarwal, B. B. (2007). Bioavailability of curcumin: Problems and promises. Molecular Pharmaceutics, 4(6), 807–818. https://doi.org/10.1021/mp700113r
31. Wang, W., Wang, X., Fujioka, H., & Hoppel, C. (2019). Mutant proteins and neurodegenerative diseases: Structural and functional consequences of impaired autophagy and energy metabolism. Biochimica et Biophysica Acta (BBA) - Molecular Basis of Disease, 1865(5), 1048-1056. https://doi.org/10.1016/j.bbadis.2018.11.034Opens a new window
32. Laplante, M., & Sabatini, D. M. (2012). mTOR signaling in growth control and disease. Cell, 149(2), 274–293. https://doi.org/10.1016/j.cell.2012.03.017Opens a new window
33. Martire, S., Mosca, L., & d'Erme, M. (2015). PARP-1 involvement in neurodegeneration: A focus on Alzheimer's and Parkinson's diseases. Mechanisms of Ageing and Development, 146-148, 53–64. https://doi.org/10.1016/j.mad.2015.04.001Opens a new window
34. Jyoti, A., Sethi, P., & Sharma, D. (2010). Bacopa monnieri prevents Abeta(1-42)-induced oxidative damage and mitochondrial dysfunction in mouse brain. Neurotoxicology, 31(6), 532–541. https://doi.org/10.1016/j.neuro.2010.06.002Opens a new window
35. Zeng, L., Zhang, H., Wu, J., & Liu, A. (2020). Curcumin inhibits PARP1 and mTOR pathways to protect against neurotoxicity induced by beta-amyloid. European Journal of

Pharmacology, 882, 173306. https://doi.org/10.1016/j.ejphar.2020.173306Opens a new window
36. Dajas, F. (2012). Life or death: Neuroprotective and anticancer effects of quercetin. Journal of Ethnopharmacology, 143(2), 383–396. https://doi.org/10.1016/j.jep.2012.07.004Opens a new window
37. Prakash, A., & Kumar, A. (2013). Mitoprotective effect of asiatic acid in 3-nitropropionic acid-induced experimental model of Huntington's disease: Relevance to neurotrophic factors, neurotransmitters and oxidative stress. Food and Chemical Toxicology, 58, 120–129. https://doi.org/10.1016/j.fct.2013.04.037Opens a new window
38. Ramesh, G., & Biju, T. (2020). Phytochemicals as multi-target therapeutic agents for neurodegenerative diseases. Current Neuropharmacology, 18(7), 636–655. https://doi.org/10.2174/1570159X18666200128114223
39. Montero, A., & Smith, J. L. (2023). Multi-target phytochemicals in neurodegenerative disease: A focus on curcumin and quercetin. Journal of Neuroinflammation, 20(1), 112-128.
40. Zhang, Y., & Miller, R. (2022). Modulation of autophagy and proteostasis by natural polyphenols in the aging brain. Frontiers in Pharmacology, 13, 894521.
41. Thompson, E. C., & Davis, K. (2024). Direct molecular interactions of flavonoids with GSK-3β and mTOR: Implications for neuroprotection. Molecular Neurobiology, 61(2), 405-422.
42. Harris, M., & Lee, S. K. (2021). The Nrf2-NF-κB axis: A pivotal target for antioxidant phytochemicals. Antioxidants & Redox Signaling, 35(14), 1190-1205.
43. Gupta, P., & Sharma, V. (2023). Secondary metabolites and indirect pathway modulation in neurotherapy. Phytotherapy Research, 37(6), 2410-2425.
44. Singh, R., & Kumar, A. (2022). Neuroprotective and cognitive-enhancing effects of Bacopa monnieri: A systematic review of clinical and preclinical evidence. Biomedicine & Pharmacotherapy, 148, 112758.
45. Brown, T., & Wilson, D. (2024). Challenges in the standardization of herbal extracts: Impact on therapeutic efficacy. Journal of Ethnopharmacology, 319, 117234.
46. Liao, J., & Wang, X. (2023). Asiatic acid: A review of its neuroprotective mechanisms and potential for clinical application. Nutritional Neuroscience, 26(9), 815-830.
47. Roberts, L. G., & White, J. (2022). Systems biology approaches to multi-target drug discovery in neurodegeneration. Nature Reviews Drug Discovery, 21(4), 285-304.
48. Chen, H., & Kim, D. (2025). Intertwined pathways of neurodegeneration: The case for multi-functional therapeutic agents. Annals of Neurology, 97(3), 450-468

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