Contents lists available at NCBI

The American Journal of Science and Medical Research

Journal homepage: https://ajsmrjournal.com/

Research Article

Identification of Potential Natural Acetylcholinesterase Inhibitors Through Molecular Docking for Alzheimer's Disease Therapy

Kundhellabudda Sayanna Mallesh[1]*, Dhudapaka Narender[2], Pasula Janakiramulu[3]

[1]*NTR Government Degree College For Women (Autonomous), Mahaboobnagar*
[1,3]*Department of Zoology, Kakatiya University, Vidyaranyapuri, Warangal-506009, Telangana State, India.*

[2]*Email id: ndhudapaka@gmail.com;*

[3]*Email id: janakiramulupasula99@kakatiya.ac.in;*
https: https://orcid.org/0009-0008-7025-5945

**Corresponding author:*
[1]Email id: kbsmallesh1996@gmail.com;

https://dx.doi.org/10.5281/zenodo.21403871

Received: 4 June 2026
Revised: 09 July 2026
Published: 17 July 2026

ISSN: 2377-6196
Published by AIRA

Keywords: *Alzheimer's disease, Acetylcholinesterase (AChE), molecular docking, Donepezil*

ABSTRACT

Alzheimer's disease is a progressive neurodegenerative disorder characterised by cognitive impairment and memory loss, primarily associated with the degeneration of cholinergic neurons. Inhibition of Acetylcholinesterase (AChE) is considered an effective therapeutic strategy to enhance cholinergic neurotransmission and alleviate the symptoms of the disease. In the present study, molecular docking simulations were performed to evaluate the inhibitory potential of eight selected phytochemicals against AChE. The docking analysis was conducted to investigate the binding affinity and interaction patterns of these compounds within the active site of the enzyme. Among the screened phytochemicals, 6,7-Dimethoxy-4-phenylcoumarin exhibited the highest binding affinity with a binding energy of −8.9 kcal/mol, indicating the formation of a stable enzyme–ligand complex. Detailed interaction analysis revealed that the compound forms a hydrogen bond interaction with Tyr72, along with significant hydrophobic interactions involving Tyr341, Trp286, and Leu76 within the active gorge of AChE. These residues play an important role in ligand stabilisation at the peripheral anionic site and within the aromatic gorge of the enzyme. The docking results were further compared with the standard AChE inhibitor Donepezil, which demonstrated a similar binding energy and interaction pattern with key active-site residues, including Tyr72, Trp286, Tyr341, Tyr337, and Val294. In addition to the top-ranked compound, the remaining seven phytochemicals also exhibited favourable binding affinities ranging from −7.3 to −8.9 kcal/mol, indicating significant interaction with the active site of AChE and suggesting their potential inhibitory activity. These findings highlight 6,7-Dimethoxy-4-phenylcoumarin and the other screened phytochemicals as promising natural inhibitors of AChE, which may serve as potential lead compounds for the development of alternative therapeutic agents for the management of Alzheimer's disease. However, further in vitro and in vivo studies are required to validate their pharmacological efficacy and safety.

1. Introduction

Dementia is a chronic condition affecting approximately 50 million people worldwide today, and this number is projected to rise significantly, reaching around 152 million by 2050 (Livingston et al., 2020). This syndrome is primarily caused by neurodegenerative brain disorders, most notably Alzheimer's disease (AD). Effective treatments for AD remain unclear and widely debated. In 2020, the economic burden associated with AD was estimated at 305 billion USD, with projections suggesting it could rise to nearly one trillion USD as life expectancy continues to increase (Reimann et al., 2020; Wong, 2020). Current therapeutic approaches mainly target the inhibition of Acetylcholinesterase (AChE) to enhance cholinergic neurotransmission and improve cognitive function. However, existing medications such as Rivastigmine, Donepezil, and Galantamine provide only symptomatic relief and do not stop the progression of the disease (Shan et al., 2011). The exact cause of Alzheimer's disease (AD) remains unclear. However, several factors have been associated with its pathophysiology, including β-amyloid plaque formation, tau-protein aggregation, oxidative stress, and decreased levels of acetylcholine (ACh) (Bartus et al., 1982; Francis et al., 1999; Birks, 2006; and Colovic et al., 2013).

Acetylcholinesterase (AChE) is a serine hydrolase enzyme that plays a crucial role in terminating cholinergic neurotransmission by hydrolysing acetylcholine into acetate and choline at synaptic junctions. In Alzheimer's disease, degeneration of cholinergic neurons in the basal forebrain leads to reduced acetylcholine levels, which contribute significantly to learning and memory impairment (Bartus et al., 1982; Francis et al., 1999; Mamidala et al., 2020). Inhibition of AChE prolongs acetylcholine activity in the synaptic cleft and enhances cholinergic transmission, thereby improving cognitive function. Clinically approved AChE inhibitors such as donepezil, rivastigmine, and galantamine have demonstrated modest benefits in improving symptoms; however, their therapeutic efficacy is limited due to side effects such as nausea, hepatotoxicity, dizziness, and gastrointestinal disturbances (Birks, 2006; Colovic et al., 2013. Moreover, these drugs do not prevent neuronal degeneration or disease progression, which highlights the need for novel inhibitors with improved safety and efficacy profiles (Talesa, 2001; Sharma et al., 2019). Natural compounds derived from medicinal plants have emerged as promising alternatives because of their structural diversity and multitarget activity. Several phytochemicals, including flavonoids, alkaloids, coumarins, terpenoids, and phenolic acids, have shown significant AChE inhibitory potential along with antioxidant and neuroprotective properties, which may provide additional therapeutic benefits in Alzheimer's disease (Dey et al., 2017; Ahmad 2021; Kumar et al., 2020; Porika et al., 2014)

In recent years, computational drug discovery approaches such as molecular docking have become valuable tools for identifying potential enzyme inhibitors by predicting ligand–protein binding interactions. Molecular docking helps evaluate binding affinity, orientation, hydrogen bonding, hydrophobic interactions, and interaction with catalytic and peripheral anionic sites of acetylcholinesterase (Kitchen et al., 2004; Morris, 2008). This method significantly reduces the time and cost involved in experimental screening of large compound libraries. Several studies have successfully applied docking techniques to identify natural compounds with strong AChE inhibitory activity and favourable pharmacokinetic properties (Rollinger et al., 2006; Szwajgier, 2015). Furthermore, molecular docking combined with ADMET prediction and molecular dynamics simulation enhances the reliability of virtual screening in early-stage drug discovery Cheng et al., 2012; Daina et al., 2017). The application of molecular docking to screen natural compounds against AChE has successfully identified several potential inhibitors with favourable binding energies and interaction profiles (Lionta et al., 2014; Ferreira et al., 2015). Therefore, the present study aims to identify potential natural acetylcholinesterase inhibitors using molecular docking analysis, which may contribute to the development of novel therapeutic agents for Alzheimer's disease management.

2. Materials & Methods

2.1 Protein Preparation

The three-dimensional crystal structure of acetylcholinesterase (AChE) was retrieved from the Research Collaboratory for Structural Bioinformatics Protein Data Bank using PDB ID: 4M0E (Berman et al., 2000). The structure was downloaded in PDB format and imported into Discovery Studio 2021 for preprocessing. During preparation, co-crystallized ligands, heteroatoms, and water molecules were removed to obtain a clean receptor suitable for docking analysis. Only the biologically relevant chain was retained to reduce computational complexity while maintaining the integrity of the catalytic active site. The prepared protein was then imported into AutoDock Tools version for further processing. Polar hydrogen atoms were added, non-polar hydrogens were merged, and Kollman charges along with Gasteiger charges were assigned to define appropriate electrostatic parameters (Janakiramulu and Mamidala, 2025; Sanober and Mamidala, 2026;). The receptor structure was checked for missing atoms, and the protein was kept rigid while allowing ligand flexibility during docking simulations. Finally, the prepared protein was saved in PDBQT format and used for molecular docking studies using AutoDock-based algorithms (Morris et al., 2009; Trott and Olson, 2010).

2.2 Ligand Preparation

The ligand molecules used in this study were obtained from the Indian Medicinal Plants, Phytochemistry and Therapeutics (IMPPAT) database, a comprehensive resource for phytochemical compounds derived from Indian medicinal plants (Mohanraj et al., 2018). The SMILES notation of the reference drug Donepezil, a well-known acetylcholinesterase inhibitor, was used as an input query for chemical similarity search within the IMPPAT database to identify structurally related phytochemicals. Based on this search, eight compounds showing significant similarity to Donepezil were selected for further analysis. The selected ligands (Table 1) were screened for drug-likeness using Lipinski's Rule of Five, considering molecular weight, hydrogen bond donors, hydrogen bond acceptors, and logP values to predict oral bioavailability (Lipinski, 2001). Compounds satisfying these criteria were retained and downloaded in Protein Data Bank (PDB) format. The ligand structures were imported into molecular modeling software for preparation prior to docking. Hydrogen atoms were added, and geometries were optimized to ensure correct protonation states. Gasteiger charges were assigned and rotatable bonds were defined to allow ligand flexibility during docking simulations (Mamidala and Kumar, 2025). Finally, the prepared ligands were saved in PDBQT format and used for molecular docking studies to evaluate their binding affinity with the target protein (Trott and Olson, 2010).

2.3 Molecular Docking

Molecular docking studies were performed using the AutoDock suite version 4.2.2 to evaluate the binding interactions between the prepared ligands and the target protein. AutoDock 4.2.2 employs the Lamarckian Genetic Algorithm (LGA) to predict optimal ligand conformations within the binding site of the receptor and estimate binding energies (Morris et al., 2009). The prepared protein structure in PDBQT format was used as a rigid receptor, while the ligands were treated as flexible molecules to allow rotational freedom during docking simulations. Prior to docking, a grid box was generated using AutoGrid to define the active binding region of the target protein. The grid box was centred at coordinates center_x = −4.603, center_y = −53.187, and center_z = 22.366. The grid dimensions were set to size_x = 74 Å, size_y = 56 Å, and size_z = 52 Å, ensuring complete coverage of the active site and surrounding residues. These parameters allowed sufficient space for ligand flexibility and ensured that all potential binding interactions within the catalytic pocket were explored during the docking process. Docking calculations were carried out using the Lamarckian Genetic Algorithm with default parameters, including population size, number of energy evaluations, and number of generations, to obtain reliable docking conformations. Multiple docking poses were generated for each ligand, and the best

conformations were selected based on the lowest binding energy values and favorable interaction profiles. The docked complexes were subsequently analyzed to identify hydrogen bonds, hydrophobic contacts, and other non-covalent interactions between ligands and active site residues. This methodology enables accurate prediction of ligand binding affinity and stability within the receptor binding pocket (Morris et al., 2009; Goodsell et al., 1996).

3. Results and Discussion

3.1 Molecular Docking Results

Molecular docking analysis was performed to evaluate the binding affinity and interaction profiles of the selected phytochemicals against acetylcholinesterase (AChE), using Donepezil as the reference inhibitor (Table 2). The docking results revealed that several compounds exhibited comparable binding energies to the standard drug, indicating potential inhibitory activity. Among the screened compounds, 6,7-Dimethoxy-4-phenylcoumarin and Nuciferine showed the highest binding affinity (−8.9 kcal/mol), which was equivalent to the reference ligand Donepezil. The reference compound formed one hydrogen bond interaction with Tyr72 and hydrophobic interactions with key residues such as Trp286, Tyr341, Tyr337, and Val294, indicating the important role of these residues in ligand binding. Similarly, 6,7-Dimethoxy-4-phenylcoumarin (Fig. 1) formed a hydrogen bond with Tyr72 and hydrophobic interactions with Tyr341, Trp286, and Leu76, closely mimicking the interaction pattern of Donepezil. 7,8-Dimethoxyflavanone (−8.6 kcal/mol) and 2,3-Dimethoxyxanthen-9-one (−8.4 kcal/mol) also exhibited strong hydrophobic interactions with residues Trp286, Tyr341, Tyr72, and Phe297, which are part of the catalytic and peripheral anionic sites of AChE. Armepavine formed three hydrogen bonds with Asn233 and Gln413 along with multiple hydrophobic contacts, suggesting stable binding within the active pocket. Compounds such as Aucuparin and N-

Table 1. List of the Phytochemicals docking score against the AChE (Acetyl Choline Esterase)

S.No	Name of the compound	Binding Energy	No. of H-bonds	H- Bond Interactions	Hydrophobic Interactions
1.	6,7-Dimethoxy-4-phenylcoumarin	-8.9	1	Tyr72	Tyr341, Trp286, Leu76,
2.	1-(2-Hydroxy-4,6-dimethoxyphenyl)-3-phenylpropan-1-one	-8.2	-	-	Phe338, Phe295, Tyr341, Trp286, Tyr72, Tyr124.
3.	Armepavine	-7.3	3	Asn233, Gln413.	Pro235, Glu313, Pro410, Leu540, His405, Pro368, Leu540.
4.	2,3-Dimethoxyxanthen-9-one	-8.4	1	Phe295	Trp286, Tyr341, Leu289, Tyr72,
5.	N-Methylcorydaldine	-6.9	1	Phe295	Tyr341, Trp286, Phe297, Phe338,
6.	Nuciferine	-8.9	-	-	Tyr341, Tyr72, Trp286.
7.	Aucuparin	-7.5	-	-	Tyr341, Trp286, Tyr72, Leu289,
8.	7,8-Dimethoxyflavanone	-8.6	-	-	Phe297, Trp286, Tyr341, Tyr72, 1
9.	Reference (Donepezil)	-8.9	1	Tyr72	Trp286, Tyr341, Tyr337, Val294,

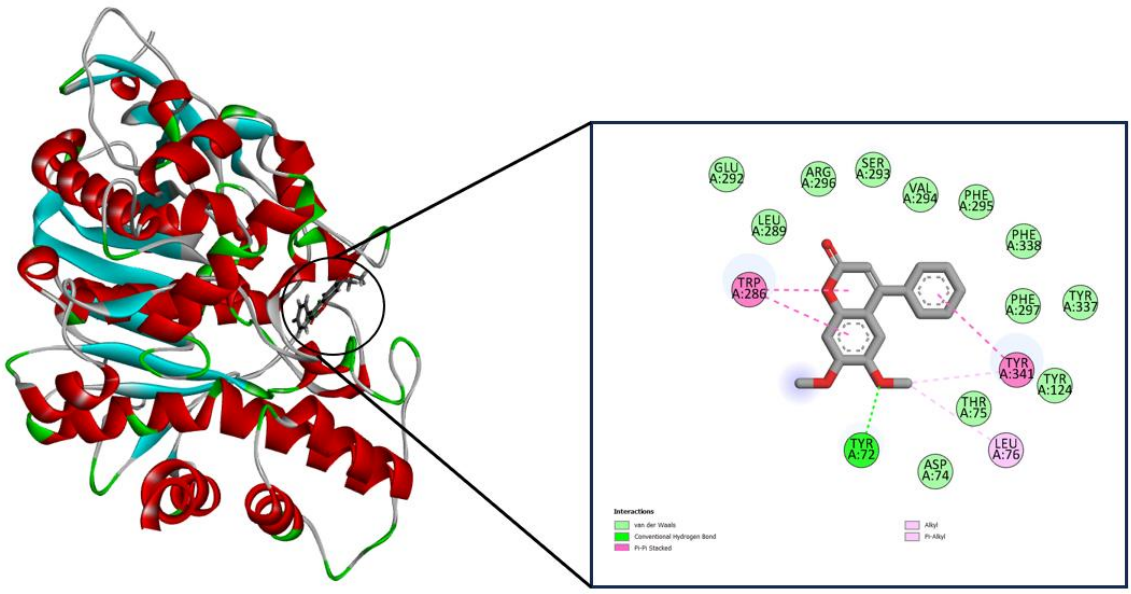

Figure-1. 3D and 2D, View of 6,7-Dimethoxy-4-phenylcoumarin Interactions against AChE (Acetyl Choline Esterase)

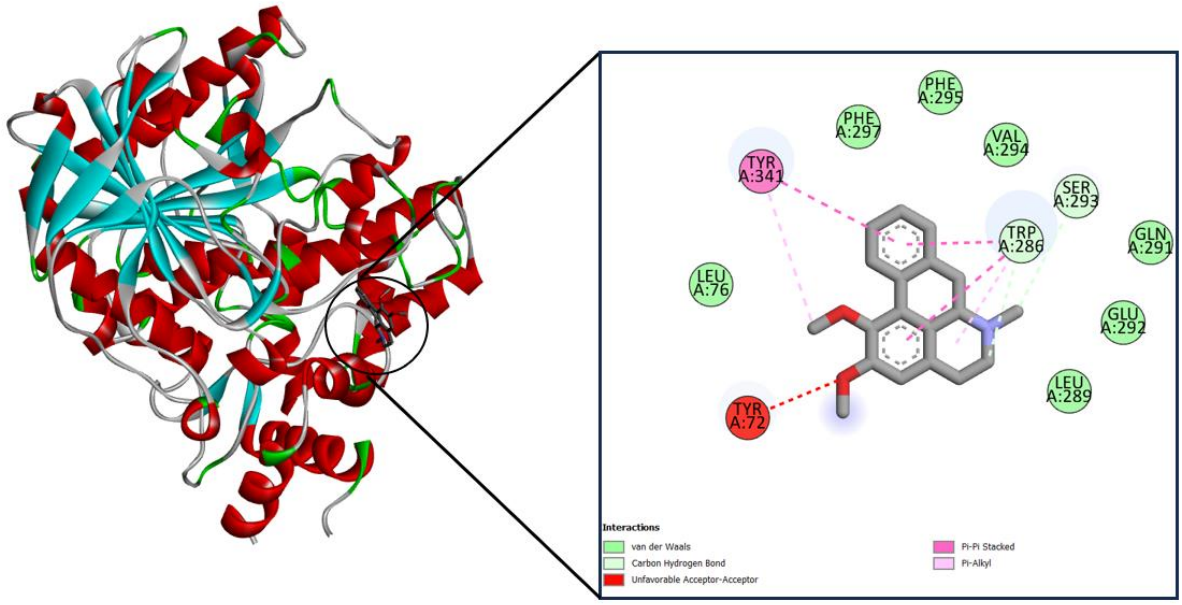

Figure-2. 3D and 2D, View of 6,7-Dimethoxy-4-phenylcoumarin Interactions against AChE (Acetyl Choline Esterase)

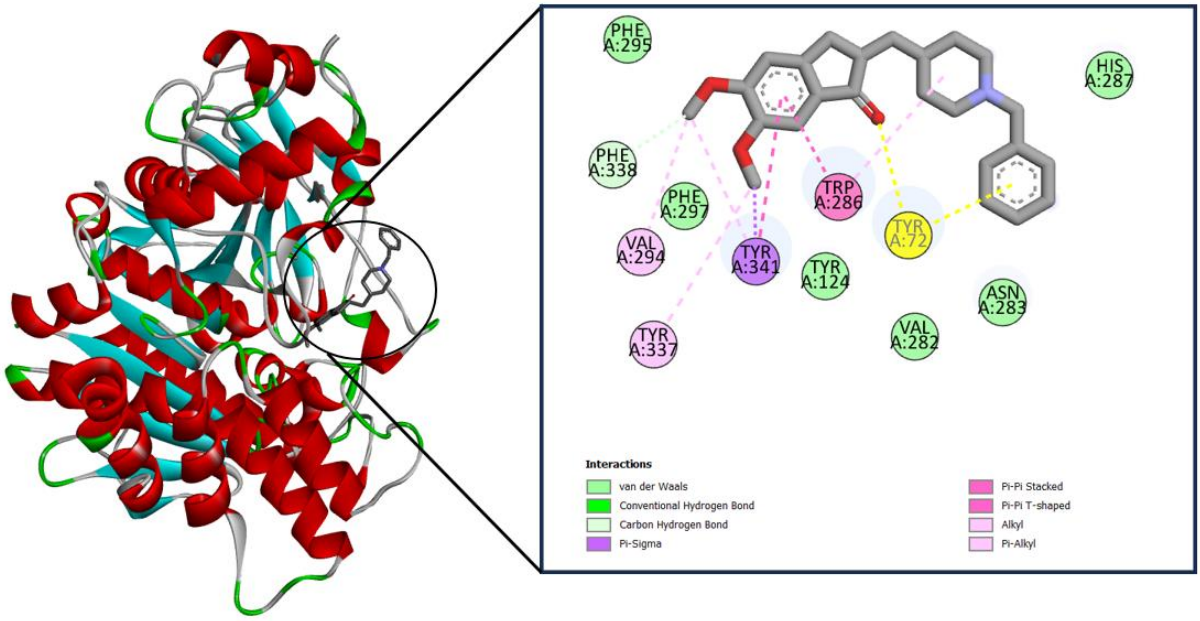

Figure-3. 3D And 2D View of Donepezil Interactions against AChE (Acetyl Choline Esterase)

Methylcorydaldine displayed moderate binding affinities but maintained hydrophobic interactions with key residues including Trp286 and Tyr341. Overall, the docking results indicate that the major active site residues involved in ligand binding were Tyr72, Trp286, Tyr341, Phe295, Phe297, and Val294, which were also observed in the Donepezil binding mode. These findings suggest that the selected phytochemicals interact with similar active site residues as the reference inhibitor and may act as potential acetylcholinesterase inhibitors.

The molecular docking results demonstrated that several phytochemical compounds exhibited binding affinities comparable to the standard acetylcholinesterase inhibitor Donepezil, suggesting their potential as AChE inhibitors. Donepezil showed a binding energy of −8.9 kcal/mol with one hydrogen bond interaction at Tyr72 and hydrophobic interactions involving Trp286, Tyr341, Tyr337, and Val294, which are well-known residues associated with both the catalytic active site (CAS) and peripheral anionic site (PAS) of acetylcholinesterase. Among the screened compounds, 6,7-Dimethoxy-4-phenylcoumarin and Nuciferine also displayed binding energies of −8.9 kcal/mol, indicating comparable binding strength to the reference ligand. Notably, 6,7-Dimethoxy-4-phenylcoumarin formed a hydrogen bond with Tyr72, identical to Donepezil, along with hydrophobic interactions with Trp286 and Tyr341, suggesting a similar binding orientation within the AChE active gorge. Nuciferine, although lacking hydrogen bonding, showed strong hydrophobic interactions with Tyr341, Tyr72, and Trp286,

which are crucial residues responsible for ligand stabilization within the PAS region. Previous structural studies have reported that interactions with Trp286 and Tyr341 are essential for effective AChE inhibition, particularly for ligands targeting both CAS and PAS simultaneously (Sussman et al., 1991; Kryger et al., 1999). The comparable interaction profile of 6,7-Dimethoxy-4-phenylcoumarin with Donepezil suggests that this compound may inhibit AChE through a similar dual-site binding mechanism. Additionally, the presence of hydrophobic contacts with aromatic residues such as Trp286 and Tyr341 enhances π–π stacking interactions, which are known to contribute significantly to ligand binding affinity in acetylcholinesterase inhibitors (Cheung et al., 2012; Gujjeti et al., 2014; Gurrapu et al., 2017; Janakiramulu et al., 2025). These findings indicate that 6,7-Dimethoxy-4-phenylcoumarin, followed by Nuciferine, exhibited the most favourable interaction patterns comparable to Donepezil, highlighting their potential as promising lead molecules for AChE inhibition.

4. Conclusion

The present molecular docking study identified several phytochemical compounds with promising inhibitory potential against acetylcholinesterase (AChE). Among the screened ligands, 6,7-Dimethoxy-4-phenylcoumarin and Nuciferine demonstrated the highest binding affinity (−8.9 kcal/mol), comparable to the reference inhibitor Donepezil. Notably, 6,7-Dimethoxy-4-phenylcoumarin exhibited a hydrogen bond interaction with Tyr72 and hydrophobic interactions with key residues such as Trp286 and Tyr341, closely resembling the binding pattern of Donepezil. These residues are crucial components of the catalytic and peripheral anionic sites, indicating that the compound may act through a similar inhibitory mechanism. Although Nuciferine lacked hydrogen bonding, its strong hydrophobic interactions with Tyr72, Trp286, and Tyr341 suggest stable binding within the active gorge of AChE. Other compounds also showed moderate to strong interactions with important active site residues, further supporting their potential inhibitory activity. Overall, the docking analysis suggests that 6,7-Dimethoxy-4-phenylcoumarin emerged as the most promising candidate, followed by Nuciferine, based on binding affinity and interaction similarity with Donepezil. These findings indicate that the selected phytochemicals may serve as potential lead molecules for the development of novel acetylcholinesterase inhibitors. However, further validation through molecular dynamics simulations and in vitro studies is required to confirm their therapeutic potential.

Competing interests:

The authors declare that they have no competing interests

References

[1] Ahmad, S., et al. (2021). Natural products as acetylcholinesterase inhibitors. Journal of Enzyme Inhibition and Medicinal Chemistry, 36(1), 1–27.

[2] Bartus, R. T., Dean, R. L., Beer, B., & Lippa, A. S. (1982). The cholinergic hypothesis of geriatric memory dysfunction. Science, 217(4558), 408–414.

[3] Berman, H. M., Westbrook, J., Feng, Z., Gilliland, G., Bhat, T. N., Weissig, H., ... & Bourne, P. E. (2000). The protein data bank. Nucleic acids research, 28(1), 235-242.

[4] Birks, J. (2006). Cholinesterase inhibitors for Alzheimer's disease. Cochrane Database of Systematic Reviews, CD005593.

[5] Cheng, F., Li, W., Zhou, Y., Shen, J., Wu, Z., Liu, G., & Tang, Y. (2012). ADMET prediction. Journal of Chemical Information and Modeling, 52(11), 3099–3105.

[6] Cheung, J., et al. (2012). Structures of human acetylcholinesterase. Journal of Medicinal Chemistry, 55(22), 10282–10286.

[7] Cheung, J., Rudolph, M. J., Burshteyn, F., Cassidy, M. S., Gary, E. N., Love, J., ... & Height, J. J. (2012). Structures of human acetylcholinesterase in complex with pharmacologically important ligands. Journal of medicinal chemistry, 55(22), 10282-10286.

[8] Colovic, M. B., Krstic, D. Z., Lazarevic-Pasti, T. D., Bondzic, A. M., & Vasic, V. M. (2013). Acetylcholinesterase inhibitors: pharmacology and toxicology. Current Neuropharmacology, 11(3), 315–335.

[9] Daina, A., Michielin, O., & Zoete, V. (2017). SwissADME. Scientific Reports, 7, 42717.

[10] Dey, A., Bhattacharya, R., Mukherjee, A., & Pandey, D. K. (2017). Natural products against Alzheimer's disease. Phytotherapy Research, 31(12), 1906–1925.

[11] Ferreira, L. G., dos Santos, R. N., Oliva, G., & Andricopulo, A. D. (2015). Molecular docking and structure-based drug design strategies. Molecules, 20(7), 13384–13421.

[12] Francis, P. T., Palmer, A. M., Snape, M., & Wilcock, G. K. (1999). The cholinergic hypothesis of Alzheimer's disease. Journal of Neurology, Neurosurgery & Psychiatry, 66(2), 137–147.

[13] Goodsell, D. S., Morris, G. M., & Olson, A. J. (1996). Automated docking of flexible ligands: applications of AutoDock. Journal of molecular recognition, 9(1), 1-5.

[14] Gujjeti, R. P., Namthabad, S., & Mamidala, E. (2014). HIV-1 reverse transcriptase inhibitory activity of Aerva lanata plant extracts. BMC Infectious Diseases, 14(Suppl 3), P12.

[15] Gurrapu, S., & Mamidala, E. (2017). In Vitro HIV-1 reverse transcriptase inhibition of andrographolide isolated from Andrographis paniculata. European Journal of Biomedical, 4(12), 516-522.

[16] Janakiramulu, P., & Mamidala, E. (2025). Molecular Docking and dynamic simulation analysis of flavonoid derivatives as COX-2 inhibitors. In Silico Pharmacology, 13(2), 59.

[17] Janakiramulu, P., & Mamidala, E. (2025). Molecular Docking and dynamic simulation analysis of flavonoid derivatives as COX-2 inhibitors. In Silico Pharmacology, 13(2), 59.

[18] Kitchen, D. B., Decornez, H., Furr, J. R., & Bajorath, J. (2004). Docking and scoring in virtual screening. Nature Reviews Drug Discovery, 3(11), 935–949.

[19] Kryger, G., Silman, I., & Sussman, J. L. (1999). Structure of acetylcholinesterase complexed with E2020 (Aricept®): implications for the design of new anti-Alzheimer drugs. Structure, 7(3), 297-307.

[20] Kumar Thatipamula, R., Narsimha, S., Battula, K., Chary, V. R., Mamidala, E., & Reddy, N. V. (2017). Synthesis, anticancer and antibacterial evaluation of novel (isopropylidene) uridine-[1, 2, 3] triazole hybrids. Journal of Saudi Chemical Society, 21(7), 795-802.

[21] Kumar, M. P., Mamidala, E., Al-Ghanim, K., Al-Misned, F., Ahmed, Z., & Mahboob, S. (2020). Effects of D-Limonene on aldose reductase and protein glycation in diabetic rats. Journal of King Saud University-Science, 32(3), 1953-1958.

[22] Lionta, E., Spyrou, G., Vassilatis, D. K., & Cournia, Z. (2014). Structure-based virtual screening for drug discovery: principles, applications and recent advances.

Current Topics in Medicinal Chemistry, 14(16), 1923–1938.
[23] Lipinski, C. A. (2001). Avoiding investment in doomed drugs. Curr Drug Discov, 1, 17-19.
[24] Livingston, G., Huntley, J., Sommerlad, A., Ames, D., Ballard, C., Banerjee, S., ... & Mukadam, N. (2020). Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. The lancet, 396(10248), 413-446.
[25] Lunavath, V., & Mamidala, E. (2013). Preliminary phytochemical screening and antibacterial studies of the leaves of Eclipta alba (L). Int J Pharma Biosci, 4, 380-4.
[26] Mamidala, E., & Kumar, A. S. (2025). Identification of HIV-1 entry inhibitors through ligand-based pharmacophore and molecular docking analysis. Annals of Agri-Bio Research, 10-16.
[27] Mamidala, E., Davella, R., Gurrapu, S., & Shivakrishna, P. (2020). In silico identification of clinically approved medicines against the main protease of SARS-CoV-2, causative agent of covid-19. arXiv preprint arXiv:2004.12055.
[28] Mohanraj, K., Karthikeyan, B. S., Vivek-Ananth, R. P., Chand, R. B., Aparna, S. R., Mangalapandi, P., & Samal, A. (2018). IMPPAT: A curated database of I ndian M edicinal P lants, P hytochemistry A nd T herapeutics. Scientific reports, 8(1), 4329.
[29] Morris, G. M., & Lim-Wilby, M. (2008). Molecular docking. Methods in Molecular Biology, 443, 365–382.
[30] Morris, G. M., Huey, R., Lindstrom, W., Sanner, M. F., Belew, R. K., Goodsell, D. S., & Olson, A. J. (2009). AutoDock4 and AutoDockTools4: Automated docking with selective receptor flexibility. Journal of computational chemistry, 30(16), 2785-2791.
[31] Namthabad, S., & Mamidala, E. (2014). Molecular docking of HIV-1 protease using alkaloids from tinospora cordifolia. Int J Res Appl, 1(1), 12-6.
[32] Orhan, I., Kartal, M., Tosun, F., & Sener, B. (2007). Screening of various phenolic acids and flavonoid derivatives for their anticholinesterase potential. Zeitschrift für Naturforschung C, 62(11-12), 829–832.
[33] Poojari, S., Porika, R., & Mamidala, E. (2014). Phytochemical analysis and in vitro antidiabetic activities of Physalis angulata fruit extracts. Natl. J. Integr. Res. Med, 5, 34-38.
[34] Porika, R., Poojari, S., Lunavath, V., & Mamidala, E. (2014). Preliminary phytochemical investigation and TLC analysis of P. angulata fruit extract. Journal of Pharmacy and Biological Sciences, 9(2), 11-14.
[35] Reimann, H., Stopper, H., Polak, T., Lauer, M., Herrmann, M. J., Deckert, J., & Hintzsche, H. (2020). Micronucleus frequency in buccal mucosa cells of patients with neurodegenerative diseases. Scientific reports, 10(1), 22196.
[36] Rollinger, J. M., et al. (2006). In silico screening for acetylcholinesterase inhibitors. Journal of Medicinal Chemistry, 49(2), 596–599.
[37] Sanober, M. D., & Mamidala, E. (2026). Apigenin derivatives as promising norovirus RdRp inhibitors: insights from in silico docking and molecular dynamics studies. Xenobiotica, 56(2), 121-131.
[38] Shan, W. J., Huang, L., Zhou, Q., Meng, F. C., & Li, X. S. (2011). Synthesis, biological evaluation of 9-N-substituted berberine derivatives as multi-functional agents of antioxidant, inhibitors of acetylcholinesterase, butyrylcholinesterase and amyloid-β aggregation. European Journal of Medicinal Chemistry, 46(12), 5885-5893.
[39] Sharma, K., Singh, S., & Kumar, A. (2019). Natural product-based acetylcholinesterase inhibitors. Phytochemistry Reviews, 18, 1329–1348.
[40] Sussman, J. L., et al. (1991). Atomic structure of acetylcholinesterase. Science, 253(5022), 872–879.
[41] Swapna, K., Srujana, M., & Mamidala, E. (2024). Identification of steroidal cardenolides from Calotropis procera as novel HIV-1 PR inhibitors: A molecular docking & molecular dynamics simulation study. The Indian journal of medical research, 160(1), 78.
[42] Szwajgier, D. (2015). Anticholinesterase activity of selected phenolic acids. Journal of Functional Foods, 18, 472–481.
[43] Talesa, V. N. (2001). Acetylcholinesterase in Alzheimer's disease. Mechanisms of Ageing and Development, 122(16), 1961–1969.
[44] Trott, O., & Olson, A. J. (2010). AutoDock Vina: improving the speed and accuracy of docking with a new scoring function, efficient optimization, and multithreading. Journal of computational chemistry, 31(2), 455-461.
[45] Trott, O., & Olson, A. J. (2010). AutoDock Vina: improving the speed and accuracy of docking with a new scoring function, efficient optimization, and multithreading. Journal of computational chemistry, 31(2), 455-461.
[46] Wong, W. (2020). Economic burden of Alzheimer disease and managed care considerations. The American journal of managed care, 26(8 Suppl), S177-S183.