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RESEARCH ARTICLE

Phytochemical profiling and neuroprotective potential of *Garcinia lanceifolia* Roxb.: *In vitro* and *in silico* insights

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Abstract

Garcinia lanceifolia Roxb. is renowned for its medicinal properties, though its neuroprotective potential remains underexplored. This study investigated the methanolic leaf extract of *G. lanceifolia* (MEGL) to identify its bioactive compounds and evaluate their neuroprotective potential through an integrated *in vitro* and *silico* approach. Gas chromatography-mass spectrometry (GC-MS) analysis identified 56 phytocompounds, with 20 exhibiting favourable pharmacokinetic properties based on Lipinskis' rule of five. *In vitro* assays revealed significant antioxidant and anti-inflammatory activities, highlighting the extracts' potential in combating oxidative stress and neuroinflammation. *In silico* analysis demonstrated strong molecular interactions of key compounds, such as precocene II, 13-hexyloxacyclotridec-10-en-2-one and methyl ricinoleate, with neuroprotective targets including IL-1α, KEAP1, serotonin, GABA and NMDA receptors. These compounds exhibited binding affinities competitive with or superior to reference drugs like ascorbic acid, diclofenac, ifenprodil, fluoxetine and diazepam. Toxicity profiling indicated minimal adverse effects, suggesting their potential for drug development. Visualizing ligand-receptor interactions provided insights into binding stability and specificity, emphasizing the therapeutic relevance of these phytocompounds. While findings are promising, further experimental validation is required to confirm their neuroprotective efficacy. This study underscores the potential of *G. lanceifolia* as a source of neuroprotective agents, paving the way for innovative treatments for neurodegenerative disorders.

Keywords: *Garcinia lanceifolia* Roxb.; GC-MS; *in silico* analysis; molecular docking; neuroprotection

Introduction

The treatment of neurological conditions has traditionally been accomplished through the use of herbal medicine. Even though the precise mechanisms by which herbal medicines exert their effects are not yet fully understood, it has been discovered that several herbal medications possess anti-inflammatory and/or antioxidant properties in various peripheral systems (1). Among the many complex mixtures of organic chemicals found in herbal products are flavonoids, sterols, alkaloids, saponins, glycosides, tannins, terpenes and fatty acids. Proponents of herbal medicines assert that a plants' medicinal potential stems from the synergistic effects of its numerous components rather than the pharmacologists' isolation of the individual chemicals of conventional medicines. Therefore, traditional medications are believed to be beneficial and have little or no adverse effects (2).

The *Garcinia* genus, part of the Clusiaceae family, has attracted considerable attention for its therapeutic potential, mainly due to its rich phytochemical composition, including flavonoids, terpenoids, alkaloids and xanthones. Notably, plants from the *Garcinia* genus have demonstrated anti-inflammatory, antioxidant and anticancer properties, yet their neuroprotective effects remain underexplored. *Garcinia lanceifolia* is a medicinal plant that has not been thoroughly studied, yet it has a variety of beneficial compounds. The species is commonly referred to as "Rupahi-thekera" in Assamese, "Pelh" in Mizo and "Rupohi tekera" in Mising. It is primarily found in the evergreen forests of Northeast India and the southern region of Bangladesh. Currently, it is at risk of becoming extinct in the environment and is commonly cultivated at home (3). Fruits, recognized for their acidity, are widely used as a raw ingredient in pickles, juice and other culinary preparations. They are employed for the treatment of dysentery and diarrhoea. *Garcinia lanceifolia* leaves have multiple uses, including being employed as a stomachic and diuretic and being cooked and consumed as vegetables (4). However, the pharmacokinetic properties of this plant, particularly in the context of neuroprotection, have not been rigorously studied.

Emerging evidence suggests that oxidative stress, neuroinflammation and excitotoxicity are critical mechanisms in the pathogenesis of neurodegenerative diseases. Key molecular targets such as interleukin-1 (IL-1α), Kelch-like ECH-associated protein 1 (KEAP1) and

neurotransmitter receptors like NMDA (N-Methyl-D-Aspartate), serotonin and Gamma-Aminobutyric Acid Type A (GABAA) receptors play significant roles in these pathological processes (5, 6). Therefore, targeting these proteins may provide novel therapeutic avenues for neuroprotection. *In silico* methods have emerged as powerful tools in drug discovery, allowing for the efficient screening and analysis of large compound libraries to predict pharmacokinetic properties, toxicity and molecular interactions. These approaches reduce the cost and time of early-stage drug discovery and prioritize compounds with the highest potential for further experimental validation (7). Given the rich phytochemical diversity of *Garcinia lanceifolia*, this study aims to explore the pharmacokinetic profiles and molecular docking interactions of its bioactive compounds with key neuroprotective targets, providing insights into their potential therapeutic applications in neurodegenerative diseases. Thus, the present study seeks to address this gap by employing a computational approach to assess the neuroprotective potential of phytocompounds identified from the methanolic leaf extract of *Garcinia lanceifolia* Roxb. This study investigates the drug-likeness, toxicity profiles and molecular interactions of these compounds with neuroprotective targets, laying the groundwork for future experimental validation and therapeutic development.

Materials and Methods

Plant material

The *Garcinia lanceifolia* Roxb leaves were collected through random sampling during April-May of 2023 from Dima Hasao district of Assam, India and were dried in the shade at room temperature. The plant was identified and authenticated at the Botanical Survey of India (BSI), Shillong, Meghalaya, with voucher no. BSI/ERC/Tech/2023-24/1266. The dried leaf samples were ground to powder form with the help of a grinder. Using methanol as a solvent in the ratio of 1:10 (w/v), the powdered leaves were extracted and filtered with the help of Whatman filter paper no. 1. The filtrate was allowed to dry in a water bath and the dried concentrated extracts were stored at 4 °C until use.

In vitro antioxidant assay: ABTS radical scavenging assay

The ABTS radical cation scavenging test evaluated the overall antioxidant activity (8). A solution of APS (2.45 mM) and ABTS (7 mM) was diluted 100X before being combined to generate ABTS radicals. 200 µl of ABTS free radical reagent and 10 µl of various stocks of standard ascorbic acid and samples were applied to 96-well plates, which were then incubated at room temperature for 10 min in the dark. Treatment-free wells were regarded as controlled. Following incubation, a microplate reader was used to measure the decolourizations' absorbance at 750 nm. The results were shown concerning the negative control. The extracts' IC50 was determined. A graph was created with the X (sample concentration) and Y (inhibition percentage relative to control) axes.

In vitro anti-inflammatory assay: Protein denaturation using bovine serum albumin (BSA)

50 µL of the sample was combined with 450 µl of BSA (for sample/STD treatment) and 450 µL of PBS (for sample/STD blank) at varying concentrations and the mixture was incubated for 20 min at 37 °C. Samples were incubated for 30 min at 70 °C after adding 150 µL of sodium phosphate buffer (pH 6.3) to each tube. Spectrophotometric measurements of turbidity were made at 660 nm. BSA was absent in the product control test; µL of PBS was used instead of extracts (9). The formula used to determine the percentage of protein denaturation inhibition is as follows in equation 1-

$$\text{Percentage inhibition} = \frac{(\text{Abs control} - \text{Abs sample})}{\text{Abs control}} \times 100$$

(Eqn. 1.)

Phytochemical profiling of methanolic leaf extracts of *Garcinia lanceifolia* Roxb. using GC-MS

GC-MS analysis of the methanolic leaf extracts of *Garcinia lanceifolia* Roxb. were performed at JNU, Delhis' Advanced Instrumentation Research Facility (AIRF) using a Shimadzu QP-2010 plus system equipped with an AOC-20i +s auto-sampler. The experiment was conducted using an RTx-5 Sil MS column. The oven temperature program was set to increase by 7 °C/min from 60 °C to 250 °C with a hold period of 3 min and by 10°C/min from 250 °C to 280 °C with a hold time of 2 min. The final temperature is held for 20 min. Maintaining a temperature of 260 °C for the injector, 0.3 µL of injected sample, a pressure of 73.3 kPa, 3.0 mL/min for purging, 16.3 mL/min for total flow, 1.21 mL/min for the column, 40.1 cm/sec for linear velocity, 10.0 for split ratio, 230 °C for the ion source, 270 °C for the interface line and m/z 40-650 for the scan mass range. The substance present was discovered using mass spectra comparison. Compounds eluted by GC-MS were analyzed and classified according to their molecular formula, structure, retention duration and peak % area.

In silico analysis

Pharmacokinetic analysis using SwissADME

To estimate the pharmacokinetic features of the compounds that were found using GC-MS, SwissADME, a web tool that offers free access to a pool of rapid yet rigorous prediction models for pharmacokinetics, physicochemical parameters, medicinal chemistry friendliness and drug-likeness, was utilized (10). To estimate physicochemical parameters, SwissADME employed multiple techniques to estimate physicochemical characteristics. These methods comprised hydrogen bond donors and acceptors, polar surface area (PSA), log P and molecular weight. To evaluate the potential drug-like qualities of the phytocompounds, Lipinskis' rule of five and drug-likeness were also taken into account. Predictions regarding gastrointestinal absorption and blood-brain barrier permeability were also made to assess the medicines' bioavailability.

Molecular docking

Ligand preparation: The ligand files were obtained in .sdf format from the PubChem database (https://pubchem.ncbi.nlm.nih.gov/) and converted to .pdbqt format using Open Babel software. The compounds were then prepared using AutoDock Tools (11).

Receptor preparation: The RCSB Protein Data Bank database obtained the target proteins' 3D X-ray crystallographic structures. The target proteins include: 1) IL-1α (PDB ID: 5UC6), the cytokine interleukin 1α, which contributes significantly to inflammatory processes (12). 2) KEAP1 (PDB ID: 2FLU), also known as Kelch-like ECH-associated protein 1, is a protein that controls steady-state levels of Nrf2 (nuclear factor erythroid 2-related factor 2) in response to oxidative stress (13). 3) NMDA receptor, glutamate-gated ion channels that are important in brain physiology and pathology (14). 4) Serotonin receptor, 5-HT1B. (PDB ID: 4IAQ) is a subtype of serotonin (5-HT) G protein-coupled receptors (GPCRs) that affect neurotransmission and control synaptic serotonin levels. It has been researched for possible therapeutic uses, especially concerning mental illnesses like depression (15, 16). 5) GABAA receptor (PDB ID: 4COF), Type-A γ-aminobutyric acid receptors (GABAARs) are the primary mediators of fast inhibitory synaptic transmission in the human brain. A decrease in GABAAR signalling has been associated with hyperactive neurological disorders such as anxiety, epilepsy and insomnia (17). The target proteins used in this study were selected for their established roles in neuroinflammation, oxidative stress and neurotransmitter signalling, which are central to neurodegenerative diseases. The AutoDock tool was used to prepare the target receptor. The non-protein part of the target proteins was removed and polar hydrogens and missing residues were added (11).

Docking: The prepared ligands and receptors were subjected to molecular docking using AutoDock Vina, a widely employed open-source molecular docking software (18). Grid box dimensions and exhaustiveness parameters were optimized to ensure reliable docking. The binding affinities of the phytocompounds were compared against those of reference drugs, including diclofenac, ascorbic acid, memantine, fluoxetine and diazepam, to provide a benchmark for neuroprotective potential.

Toxicity profiling: The toxicity assessment of the phytocompounds was conducted using a web-based tool known as ProTox 3.0. ProTox 3.0 serves as a virtual laboratory accessible to academic and non-commercial users via a web server, specializing in predicting various toxicological endpoints associated with chemical structures. ProTox 3 employs computational models trained on authentic *in vitro* and *in vivo* data from well-established databases, including SuperToxic for acute toxicity, ChEMBL for cardiotoxicity and Novartis *in vitro* safety panels for toxicity target prediction. Additionally, datasets from Tox21, ClinTox and EFSAs' OpenFoodTox, as well as curated literature and publicly available toxicological databases, contribute to its robust predictive framework. These diverse sources ensure reliable toxicity predictions across multiple endpoints (19). Toxicity endpoints like hepatotoxicity, neurotoxicity, nephrotoxicity, immunotoxicity, carcinogenicity, cardiotoxicity and cytotoxicity of the phytocompounds were assessed and recorded.

Visualization: The interaction of the shortlisted ligand-protein complexes that showed the best binding affinities was visualized using Pymol and Dassault Systemes' BIOVIA Discovery Studio Visualizer. Type of interactions, amino acid residues and bond distance were visualized.

Results

In vitro antioxidant assay: ABTS radical scavenging assay

The ABTS radical scavenging assay revealed that the methanolic extract of *Garcinia lanceifolia* leaves (MEGL) exhibits notable antioxidant potential, with an IC50 value of 23.32 µg/mL. This is comparable to the standard ascorbic acid, which displayed an IC50 value of 16.67 µg/mL. Although slightly less effective than the standard (Fig. 1A), the extract demonstrates significant radical scavenging activity, underscoring its potential as a natural antioxidant source. These findings suggest that MEGL could play a pivotal role in combating oxidative stress and warrant further investigation to isolate and characterize its bioactive constituents.

In vitro anti-inflammatory assay: Protein denaturation using bovine serum albumin (BSA)

The anti-inflammatory activity of the methanolic extract of *Garcinia lanceifolia* leaves (MEGL) was evaluated and compared with the standard diclofenac. The IC50 value of MEGL was found to be 327.99 µg/mL, which is moderately higher than diclofenacs' IC50 value of 278.09 µg/mL, indicating a slightly reduced efficacy (Fig 1B). However, the results highlight the extracts' promising anti-inflammatory activity, suggesting its potential as a natural alternative. These findings emphasize the need for further studies to identify the active phytocompounds and their mechanisms of action.

Phytochemical profiling using GC-MS

56 compounds were identified from the methanolic leaf extract of *Garcinia lanceifolia* Roxb. via GC-MS analysis (Table 1). These compounds included various flavonoids, terpenoids, fatty acids and sterols, many of which have been previously reported for their biological activities.

In silico analysis

Pharmacokinetic analysis using SwissADME

Based on Lipinskis' rule of five, pharmacokinetic profiling revealed that 20 of the 56 identified compounds met the criteria for drug-likeness. These compounds demonstrated favorable physicochemical properties such as appropriate molecular weight (<500 Da), hydrogen bond donors and acceptors and LogP values within the optimal range (Table 2).

Molecular docking results

Out of the 20 compounds that met the criteria for drug-likeness, 2H-1-benzopyran, 6,7-dimethoxy-2,2-dimethyl- (precocene II), methyl (9Z)-12-hydroxy-9-octadecenoate (methyl ricinoleate), 13-hexyloxacyclotridec-10-en-2-one showed the best binding affinities against the selected

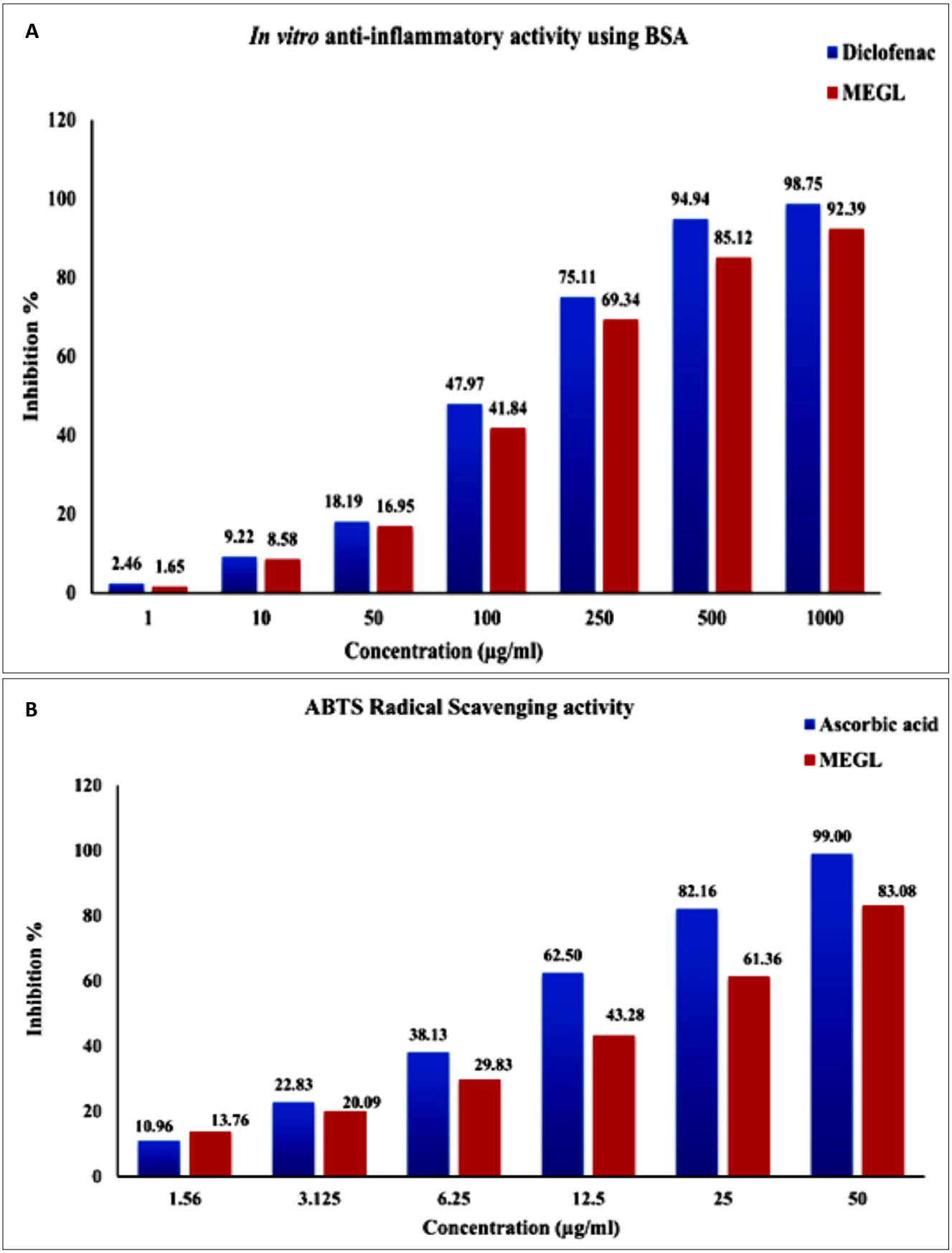

Fig. 1. A) Effect of MEGL on ABTS radical scavenging capacity. MEGL showed dose-dependent antioxidant activity with an IC50 of 23.32 µg/mL, indicating notable efficacy compared to ascorbic acid (IC50 = 16.67 µg/mL); B) Effect of MEGL on protein denaturation (Bovine Serum Albumin). MEGL demonstrated inhibitory activity against BSA denaturation, suggesting potential anti-inflammatory properties.

Table 1. GC-MS identified compounds

Peak#	R. Time	Area %	Name of compounds
1	12.044	2.33	2-[(Trimethylsilyl)oxy]propan-1-ol
2	12.683	0.15	Nonanoic acid, TMS derivative
3	12.996	0.3	2-[(Trimethylsilyl)oxy]propan-1-ol
4	13.07	0.21	Naphthalene, 1,2,3,4,4a,7-hexahydro-1,6-dimethyl-4-(1-methylethyl)-
5	14.175	0.17	Benzo[c]furanone, 3,3,4,7-tetramethyl-
6	15.118	0.2	2(4H)-Benzofuranone, 5,6,7,7a-tetrahydro-4,4,7a-trimethyl-, (R)-
7	15.227	0.11	9,12-Octadecadienoic acid (Z,Z)-, 2-[(Trimethylsilyl)oxy]-1-[[(trimethylsilyl)oxy]methyl]ethyl ester
8	15.302	0.89	2-Buten-1-ol, 2-ethyl-4-(2,2,3-trimethyl-3-cyclopenten-1-yl)-
9	15.825	0.77	2,2,18,18-Tetramethyl-3,6,10,13,17-pentaoxa-2,18-disilaneonadecane
10	16.053	0.19	Pregnane-3,20-diol, (3.alpha.,5.beta.,20S)-, 2TMS derivative
11	16.33	0.24	1,2,3-Butanetriol, 3TMS derivative
12	16.5	1.19	2H-1-Benzopyran, 6,7-dimethoxy-2,2-dimethyl-
13	16.674	0.6	1-Naphthalenol, decahydro-1,4a-dimethyl-7-(1-methylethylidene)-, [1R-(1.alpha.,4a.beta.,8a.alpha.)]-
14	17.651	0.37	Tetradecanoic acid
15	17.903	0.14	2(4H)-Benzofuranone, 5,6,7,7A-tetrahydro-6-hydroxy-4,4,7A-trimethyl-, (6S-CIS)-
16	18.428	0.74	Neophytadiene
17	18.492	2.48	2-Pentadecanone, 6,10,14-trimethyl-
18	18.628	0.12	4,6,6,7,8,8-Hexamethyl-1,3,4,6,7,8-hexahydrocyclopenta[G]isochromene
19	18.878	0.33	2-Hexadecen-1-ol, 3,7,11,15-tetramethyl-, [R-[R*,R*-(E)]]-
20	19.329	1.17	Hexadecanoic acid, methyl ester
21	19.843	7.98	n-Hexadecanoic acid
22	20.505	12.8	Palmitic Acid, TMS derivative
23	20.697	0.09	Tetradecanoic acid
24	20.794	0.42	13-Hexyloxacyclotridec-10-en-2-one
25	20.977	0.58	9,12-Octadecadienoic acid, methyl ester
26	21.04	1.3	9-Octadecenoic acid (Z)-, methyl ester
27	21.147	0.96	Phytol
28	21.268	0.26	Methyl stearate
29	21.561	11.86	9-Octadecenoic acid (Z)-
30	21.672	2.29	Phytol, TMS derivative
31	22.083	2.52	9-Octadecenoic acid, (E)-, TMS derivative
32	22.272	0.82	Octadecanoic acid, trimethylsilyl ester
33	22.781	1.43	Methyl (9Z)-12-hydroxy-9-octadecenoate #
34	23.17	0.12	Ricinoleic acid, 2TMS derivative
35	23.291	0.86	4,8,12,16-Tetramethylheptadecan-4-olide
36	23.657	0.61	1-Decanol, 9-[(trimethylsilyl)oxy]-, trifluoroacetate
37	24.503	0.83	Methyl dihydomalvalate
38	24.604	0.86	Hexadecanoic acid, 2-hydroxy-1-(hydroxymethyl)ethyl ester
39	24.936	0.96	1-Monopalmitin, 2TMS derivative
40	25.323	1.4	13-Docosenoic acid, (Z)-, TMS derivative
41	25.484	0.26	Behenic acid, TMS derivative
42	26.008	4.5	9-Octadecenoic acid (Z)-, 2,3-dihydroxypropyl ester
43	26.24	3.11	9,12-Octadecadienoic acid (Z,Z)-, 2-[(trimethylsilyl)oxy]-1-[[(trimethylsilyl)oxy]methyl]ethyl ester
44	26.603	0.25	d-Glucosamine
45	26.827	3.46	Squalene
46	27.459	0.54	Ricinoleic acid, 2TMS derivative
47	27.639	1.5	2-Oleoylglycerol, 2TMS derivative
48	29.09	0.87	Stigmasta-5,22-dien-3-ol, acetate, (3.beta.)-
49	29.295	0.85	.beta.-Sitosterol acetate
50	29.486	1.88	2H-1-benzopyran-6-ol, 3,4-duhydro-2,5,7,8-tetramethyl-2-(4,8,12-trimethyltridecyl)-, [2R-[2R*(4R*,8R*)]]-
51	31.93	4.87	Ergostane-3,12-diol, (3.alpha.,5.beta.,12.alpha.)-
52	32.879	9.65	4,22-Stigmastadiene-3-one
53	33.194	3.98	9,19-Cyclolanost-24-en-3-ol, (3.beta.)-
54	33.371	0.89	5H-3,5a-Epoxynaphth[2,1-c]oxepin, dodecahydro-3,8,8,11a-tetramethyl-, [3S-(3.alpha.,5a.alpha.,7a.alpha.,11a.beta.,11b.alpha.)]-
55	33.751	0.46	Thunbergol
56	33.959	1.28	Pregn-4-ene-3,20-dione

Table 2. Pharmacokinetic properties of GC-MS identified compounds

	Compounds	MW	HBA	HBD	MR	iLOGP	TPSA	GI absorption	BBB permeant	Lipinski #violations
1	beta.-Sitosterol acetate	456.74	2	0	142.97	5.19	26.3	Low	No	1
2	1,2,3-Butanetriol, 3TMS derivative	322.66	3	0	91.46	4.49	27.69	High	Yes	0
3	13-Docosenoic acid, (Z)-, TMS derivative	410.75	2	0	130.97	6.24	26.3	Low	No	1
4	13-Hexyloxacyclotridec-10-en-2-one	280.45	2	0	87.34	4.03	26.3	High	Yes	1
5	1-Decanol, 9-[(trimethylsilyl)oxy]-, trifluoroacetate	342.47	6	0	84.65	4.58	35.53	High	No	0
6	1-Monopalmitin, 2TMS derivative	474.86	4	0	141.48	7.13	44.76	Low	No	1
7	1-Naphthalenol, decahydro-1,4a-dimethyl-7-(1-methylethylidene)-, [1R-(1.alpha.,4a.beta.,8a.alpha.)]-	222.37	1	1	70.46	3.1	20.23	High	Yes	0
8	Loliolide	180.24	2	0	51.35	2.29	26.3	High	Yes	0
9	2(4H)-benzofuranone, 5,6,7,7A-tetrahydro-6-hydroxy-4,4,7A-trimethyl-, (6S-cis)-	196.24	3	1	52.51	1.88	46.53	High	Yes	0
10	2,2,18,18-Tetramethyl-3,6,10,13,17-pentaoxa-2,18-disilaneonadecane	366.64	5	1	100.6	5.11	57.15	High	Yes	0
11	2-[(Trimethylsilyl)oxy]propan-1-ol	148.28	2	1	41.07	2.38	29.46	High	Yes	0
12	2-[(Trimethylsilyl)oxy]propan-1-ol	148.28	2	1	41.07	2.38	29.46	High	Yes	0
13	2-Buten-1-ol, 2-ethyl-4-(2,2,3-trimethyl-3-cyclopenten-1-yl)-	208.34	1	1	67.25	3.15	20.23	High	Yes	0
14	2H-1-Benzopyran, 6,7-dimethoxy-2,2-dimethyl-	220.26	3	0	63.55	2.96	27.69	High	Yes	0
15	2H-1-benzopyran-6-ol, 3,4-dihydro-2,5,7,8-tetramethyl-2-(4,8,12-trimethyltridecyl)-, [2R-[2R*(4R*,8R*)]]-	430.71	2	1	139.27	5.92	29.46	Low	No	1
16	2-Hexadecen-1-ol, 3,7,11,15-tetramethyl-, [R-[R*,R*-(E)]]-	296.53	1	1	98.94	4.71	20.23	Low	No	1
17	2-Oleoylglycerol, 2TMS derivative	500.9	4	0	150.62	7.44	44.76	Low	No	2
18	2-Pentadecanone, 6,10,14-trimethyl-	268.48	1	0	88.84	4.39	17.07	High	No	1
19	4,22-Stigmastadiene-3-one	410.67	1	0	131.79	4.67	17.07	Low	No	1
20	Galaxolide	258.4	1	0	81.39	3.47	9.23	High	Yes	1
21	4,8,12,16-Tetramethylheptadecan-4-olide	324.54	2	0	102.27	4.15	26.3	Low	No	1
22	5H-3,5a-Epoxynaphth[2,1-c]oxepin, dodecahydro-3,8,8,11a-tetramethyl-	278.43	2	0	81.91	3.52	18.46	High	Yes	0
23	9,12-octadecadienoic acid (Z,Z)-, 2-[(trimethylsilyl)oxy]-1-[[(trimethylsilyl)oxy]methyl]ethyl ester	498.89	4	0	150.15	7.32	44.76	Low	No	1
24	9,12-Octadecadienoic acid (Z,Z)-, 2-[(trimethylsilyl)oxy]-1-[[(trimethylsilyl)oxy]methyl]ethyl ester	498.89	4	0	150.15	7.32	44.76	Low	No	1
25	9,12-Octadecadienoic acid, methyl ester	294.47	2	0	93.78	4.61	26.3	High	No	1
26	9,19-Cyclolanost-24-en-3-ol, (3.beta.)-	426.72	1	1	135.14	5.17	20.23	Low	No	1
27	9-Octadecenoic acid (Z)-	282.46	2	1	89.94	4.27	37.3	High	No	1
28	9-Octadecenoic acid (Z)-, 2,3-dihydroxypropyl ester	356.54	4	2	106.2	4.33	66.76	High	Yes	0
29	9-Octadecenoic acid (Z)-, methyl ester	296.49	2	0	94.26	4.75	26.3	High	No	1
30	9-Octadecenoic acid, (E)-, TMS derivative	354.64	2	0	111.74	5.84	26.3	Low	No	1

31	Behenic acid, TMS derivative	412.76	2	0	131.44	6.94	26.3	Low	No	1
32	Benzo[c]furanone, 3,3,4,7-tetramethyl-	190.24	2	0	55.2	2.38	26.3	High	Yes	0
33	d-Glucosamine	179.17	6	5	37.28	0.21	116.17	Low	No	0
34	Ergostane-3,12-diol, (3.alpha.,5.beta.,12.alpha.)-	418.7	2	2	130.06	4.66	40.46	High	No	1
35	Hexadecanoic acid, 2-hydroxy-1-(hydroxymethyl)ethyl ester	330.5	4	2	97.06	4.5	66.76	High	Yes	0
36	Hexadecanoic acid, methyl ester	270.45	2	0	85.12	4.41	26.3	High	Yes	1
37	Methyl (9Z)-12-hydroxy-9-octadecenoate #	312.49	3	1	95.42	4.45	46.53	High	Yes	0
38	Methyl dihydromalvalate	296.49	2	0	92.62	4.5	26.3	High	No	1
39	Methyl stearate	298.5	2	0	94.73	4.81	26.3	High	No	1
40	Naphthalene, 1,2,3,4,4a,7-hexahydro-1,6-dimethyl-4-(1-methylethyl)-	204.35	0	0	69.04	3.39	0	Low	No	1
41	Neophytadiene	278.52	0	0	97.31	5.05	0	Low	No	1
42	n-Hexadecanoic acid	256.42	2	1	80.8	3.85	37.3	High	Yes	1
43	Nonanoic acid, TMS derivative	230.42	2	0	68.95	3.79	26.3	High	Yes	0
44	Octadecanoic acid, trimethylsilyl ester	356.66	2	0	112.21	6.01	26.3	Low	No	1
45	Palmitic Acid, TMS derivative	328.61	2	0	102.6	5.33	26.3	Low	No	1
46	Phytol	296.53	1	1	98.94	4.71	20.23	Low	No	1
47	Phytol, TMS derivative	368.71	1	0	121.15	6.16	9.23	Low	No	1
48	Progesterone	314.46	2	0	94.01	3.08	34.14	High	Yes	0
49	Pregnane-3,20-diol, (3.alpha.,5.beta.,20S)-, 2TMS derivative	464.87	2	0	140.83	5.81	18.46	Low	No	1
50	Ricinoleic acid, 2TMS derivative	442.82	3	0	135.11	6.68	35.53	Low	No	1
51	Ricinoleic acid, 2TMS derivative	442.82	3	0	135.11	6.68	35.53	Low	No	1
52	Squalene	410.72	0	0	143.48	6.37	0	Low	No	1
53	Stigmasta-5,22-dien-3-ol, acetate, (3.beta.)-	454.73	2	0	142.49	5.2	26.3	Low	No	1
54	Tetradecanoic acid	228.37	2	1	71.18	3.32	37.3	High	Yes	0
55	Tetradecanoic acid	228.37	2	1	71.18	3.32	37.3	High	Yes	0
56	Thunbergol	290.48	1	1	95.92	3.85	20.23	High	No	1

*MW-Molecular weight; HBA- Hydrogen Bond Acceptor; HBD-Hydrogen Bond Donor; MR-Molar Refractivity; TPSA-Topological Polar Surface Area; GI-Gastrointestinal; BBB-Blood Brain Barrier

target proteins. Precocene II gave the best docking score of -5.8 kcal/mol, slightly less than the reference compound diclofenac (-6.2 kcal/mol) when docked against IL-1α. Precocene II also gave the best binding affinity (-7.3 kcal/mol) against KEAP1 compared to the reference compound ascorbic acid (-6.6 kcal/mol). Against the NMDA receptor, methyl ricinoleate showed the best binding affinity (-6.7 kcal/mol) among all compounds, although slightly lesser than the reference compound ifenprodil (-7.8 kcal/mol). Of all the compounds, 13-hexyloxacyclotridec-10-en-2-one showed the best docking score (-7.7 kcal/mol) against serotonin receptor concerning the reference compound, fluoxetine (-8.1 kcal/mol). 13-Hexyloxacyclotridec-10-en-2-one also showed the best binding affinity (-7.2 kcal/mol) against GABAA receptor with respect to the reference compound diazepam (-7.9 kcal/mol).

ProTox 3.0 analysis

The best-docked compounds were evaluated for toxicity using ProTox 3.0. The predicted toxicity of the 3 compounds showed inactivity at all toxicity endpoints except for precocene II, which showed activity in carcinogenicity and immunogenicity (Table 3).

Visualization

The visualization of molecular interactions between these target receptors and the phytocompounds in Fig. 2A—E reveals various binding interactions, including conventional hydrogen bonds, carbon-hydrogen bonds, alkyl and pi-alkyl interactions with the amino acid residues of the receptors. Table 4 presents the interactions between the best-docked phytocompounds and the amino acid residues within the active sites of five target proteins, namely IL-1α, KEAP1, NMDA receptor, serotonin receptor and GABAA receptor. Each compound is shown to bind with specific amino acid residues in these active sites with bond lengths ranging from 1.89 to 5.45 Å, which suggests their potential role in modulating the activities of the target proteins.

Discussion

According to research, the bark of *Garcinia lanceifolia* Roxb. has a dose-dependent antiulcer effect and significant effects on antioxidant enzymes, blood glucose levels, lipid profiles and histopathological studies. Additionally, the

Table 3. Toxicity profiling of the best-docked phytocompounds

Sl.no.	Compounds	Toxicity endpoints						
		Hepatotoxicity	Neurotoxicity	Nephrotoxicity	Cardiotoxicity	Carcinogenicity	Immunotoxicity	Cytotoxicity
1	13-Hexyloxacyclotridec-10-en-2-one	Inactive	Inactive	Inactive	Inactive	Inactive	Inactive	Inactive
2	Methyl (9Z)-12-hydroxy-9-octadecenoate (Methyl ricinoleate)	Inactive	Inactive	Inactive	Inactive	Inactive	Inactive	Inactive
3	2H-1-benzopyran, 6,7-dimethoxy-2,2-dimethyl- (Precocene II)	Inactive	Inactive	Inactive	Inactive	Active	Active	Inactive

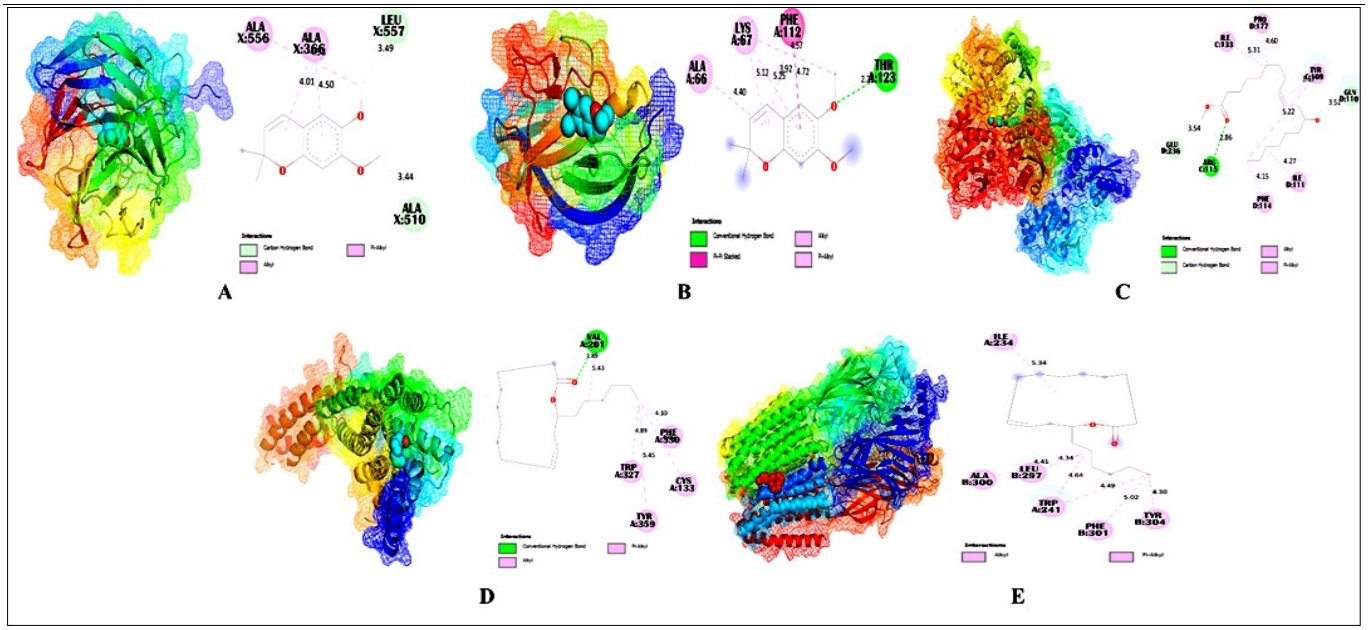

Fig. 2. 3D and 2D structures of ligand-protein complexes (Best-docked compound with their target proteins). A) Precocene II docked with KEAP1; B) Precocene II docked with IL-1α; C) Methyl ricinoleate docked with NMDA receptor; D) 13-Hexyloxacyclotridec-10-en-2-one docked with Serotonin receptor; E) 13-Hexyloxacyclotridec-10-en-2-one docked with GABAA receptor. Both 3D binding poses and 2D interaction diagrams illustrate key molecular interactions stabilizing each complex.

Table 4. Active sites and amino acid residues of target receptors with their best-docked phytocompounds

Ligand-receptor complex	Active sites & amino acid residues	Type of interactions
Precocene II-KEAP1	(Ala366, Ala510 Ala556, Leu557) of A chain	carbon-hydrogen bond, pi-alkyl, alkyl
Precocene II-IL-1α	(Ala66, Lys67, Phe112, Thre123) of A chain	conventional hydrogen bond, pi-pi stacked, pi-alkyl, alkyl
Methyl ricinoleate-NMDA receptor	(Tyr109, Arg115, Ile133) of C chain; (Gln110, Ile111, Phe114, Arg115, Pro177, Glu236) of D chain	conventional hydrogen bond, carbon-hydrogen bond, pi-alkyl, alkyl
13-Hexyloxacyclotridec-10-en-2-one-Serotonin receptor	(Val201, Phe330, Trp327, Cys133, Tyr359) of A chain	conventional hydrogen bond, pi-alkyl, alkyl
13-Hexyloxacyclotridec-10-en-2-one-GABAA receptor	(Ile234, Trp241) of A chain; (Leu297, Ala300, Phe301, Tyr304) of B chain)	pi-alkyl, alkyl

LD50 of the hydroalcoholic extract of this plant was determined to be greater than 5000 mg/kg body weight, indicating its safety profile (20). In addition, research has assessed its potential in managing pain and hyperglycemia by examining its antinociceptive, antihyperglycemic and membrane-stabilizing properties (21). Despite these findings, the neuroprotective potential of *Garcinia lanceifolia* remains largely unexamined.

The ABTS assay showed MEGLs' antioxidant activity with an IC50 of 23.32 µg/mL, slightly less effective than ascorbic acid (IC50 = 16.67 µg/ml). This significant radical scavenging activity suggests its potential to combat oxidative stress, likely due to bioactive phytochemicals like polyphenols, flavonoids and alkaloids (4). MEGL also demonstrated anti-inflammatory activity with an IC50 of 327.99 µg/mL, moderately higher than diclofenac (IC50 = 278.09 µg/mL). While less potent, it still shows promise as a natural alternative for managing inflammation. These findings underscore MEGLs' potential as a source of natural antioxidants and anti-inflammatory agents, warranting further studies to identify its active compounds and mechanisms of action.

The *in silico* findings of this study provide compelling evidence for the neuroprotective potential of phytocompounds derived from *Garcinia lanceifolia* Roxb., specifically 2H-1-benzopyran, 6,7-dimethoxy-2,2-dimethyl (precocene II), 13-hexyloxacyclotridec-10-en-2-one and methyl (9Z)-12-hydroxy-9-octadecenoate (methyl ricinoleate). These compounds exhibited favourable pharmacokinetic profiles, low toxicity and strong binding affinities to key neuroprotective targets, suggesting their potential as therapeutic agents for neurodegenerative

diseases. The pharmacokinetic analysis revealed that the selected compounds possess drug-like characteristics, particularly regarding GI absorption and BBB permeability. These are critical factors for developing neuroprotective agents, as the ability to cross the blood-brain barrier is essential for targeting central nervous system (CNS) disorders. The high BBB permeability scores of precocene II, 13-hexyloxa-cyclotridec-10-en-2-one and methyl ricinoleate, as predicted by *in silico* modelling, suggest that these compounds may readily reach the CNS, enhancing their therapeutic potential. Toxicity profiling using ProTox 3.0 further supported the suitability of these compounds for drug development. The absence of hepatotoxicity and nephrotoxicity in the top candidates reduces the risk of adverse effects, a crucial consideration in early-stage drug discovery.

The molecular docking studys' findings highlight the potential of these phytocompounds as competitive alternatives or adjuncts to existing neuroprotective agents, underscoring their relevance and efficacy in neuroprotection. Precocene II demonstrated superior binding affinity when docked with IL-1α and KEAP1 compared to other GC-MS-identified compounds. Despite its predicted toxicity profile indicating activity in carcinogenicity and immunotoxicity, precocene II, a chromene compound derived from *Ageratum conyzoides*, exhibits a range of medicinal properties primarily due to its biological activities. Notably, it has demonstrated significant antioxidant activity. In a study involving methyl jasmonate-elicited shoot cultures of *Ageratum conyzoides*, precocene II showed enhanced antioxidant enzyme levels and *in vitro* antioxidant activity (22).

Methyl ricinoleate showed the best docking score against the ligand-binding domain of the NMDA receptor, explicitly interacting with chain A, suggesting strong potential as a neuroprotective agent. However, research on its neuroprotective effects is limited and its interaction with the NMDA receptor remains unexplored. It is a derivative of ricinoleic acid, a compound known for its anti -inflammatory properties, potentially modulating pathways involved in oxidative stress and inflammation, key factors in neurodegeneration (23, 24). Its superior oxidative stability compared to methyl oleate also enhances its suitability for pharmaceutical use, potentially improving efficacy in counteracting neuronal damage (25). Despite these promising attributes, the lack of direct studies on methyl ricinoleates' neuroprotective mechanisms highlights a significant research gap. Future studies are essential to validate its role in neuroprotection, especially in conditions linked to NMDA receptor dysfunction, such as Alzheimers' and ischemic brain injury.

13-Hexyloxacyclotridec-10-en-2-one demonstrated the best docking affinity with Serotonin and GABAA receptors, suggesting its potential as a neuroprotective agent. However, there is limited research explicitly addressing its role as a neuroprotectant. This compound, a lactone derivative identified in *Ambrosia maritima*, belongs to a class of compounds renowned for their pharmacological activities, including neuroprotection. Lactones, particularly sesquiterpene lactones, have been extensively documented for their role in combating neurodegenerative processes. Notably, these compounds exhibit a broad spectrum of activities, such as antioxidant, anti-inflammatory and anti-amyloid effects, which are highly relevant to the pathophysiology of neurodegenerative diseases like Alzheimers' and Parkinsons' (26). Despite the promising docking results, further studies are needed to explore the specific neuroprotective effects of 13-hexyloxacyclotridec-10-en-2-one and its mechanisms of action.

The visualization of molecular interactions between the target receptors and the identified phytocompounds reveals a diverse array of bonding types that contribute to the stability and specificity of the binding. The interaction profile between the best-docked compounds and target proteins revealed conventional hydrogen bonds, π-alkyl, carbon-hydrogen bonds, alkyl and π-π stacking interactions. Conventional hydrogen bonds typically involve electronegative atoms like oxygen or nitrogen, significantly stabilizing ligand-receptor interactions by forming strong directional bonds (27). Unconventional hydrogen bonds, such as C-H.π and C-H.O, also contribute to molecular recognition and stability despite being weaker than conventional hydrogen bonds (28). π-π stacking interactions, characterized by the overlap of π-electron clouds, play a role in molecular recognition and stabilize the ligand-receptor complex (29).

Additionally, alkyl interactions involving nonpolar alkyl groups play a significant role in the hydrophobic effect, a major driving force in stabilizing ligand-receptor complexes. These interactions are particularly critical in environments where water is displaced by the ligand (27). Understanding these interactions is crucial for rational drug design, as they influence binding affinity and specificity (30)

Despite the promising findings, several limitations of this study should be acknowledged. First, while *in silico* methods provide valuable preliminary insights, experimental validation is required to confirm the bioactivity of these compounds. Furthermore, *in vivo* studies using animal models of neurodegeneration will be crucial to establish the efficacy and safety of these compounds in a physiological context.

Conclusion

In conclusion, our comprehensive computational approach allowed us to screen, prioritize and elucidate the pharmacokinetic properties, toxicological profiles and molecular interactions of phytocompounds from *Garcinia lanceifolia* Roxb. This study offers a strong foundation for future experimental validation and the development of innovative treatments for neurodegenerative diseases. Additionally, our findings offer valuable insights into the potential neuroprotective benefits of these compounds, encouraging further research in this domain.

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Authors' contributions

LB carried out the entire study. MB conceived of the study and participated in its design and coordination. All authors read and approved the final manuscript.

Compliance with ethical standards

Conflict of interest: Authors do not have any conflict of interests to declare.

Ethical issues: None

References

1. Barbalho SM, Leme Boaro B, da Silva Camarinha Oliveira J, Patočka J, Barbalho Lamas C, Tanaka M, et al. Molecular Mechanisms Underlying Neuroinflammation Intervention with Medicinal Plants: A Critical and Narrative Review of the Current Literature. Pharmaceuticals. 2025;18(133):1–28. https://doi.org/10.3390/ph18010133
2. Kumar GP, Khanum F. Neuroprotective potential of phytochemicals. Pharmacogn Rev. 2012;6(12):81–90. https://doi.org/10.4103/0973-7847.99898
3. Chowdhury T, Handique PJ. Evaluation of antibacterial activity and phytochemical activity of *Garcinia lancifolia* Roxb. Int J Pharm Sci Res. 2012;3(6):1663–67.
4. Policegoudra RS, Saikia S, Das J, Chattopadhyay P, Singh L, Veer V. Phenolic Content, Antioxidant Activity, Antibacterial Activity and Phytochemical Composition of *Garcinia lancifolia*. Ind J Pharm Sci. 2012;74(3):268–71. https://doi.org/10.4103/0250-474X.106075
5. Karvandi MS, Sheikhzadeh Hesari F, Aref AR, Mahdavi M. The neuroprotective effects of targeting key factors of neuronal cell death in neurodegenerative diseases: The role of ER stress, oxidative stress and neuroinflammation. Front Cell Neurosci. 2023;17:1–24. https://doi.org/10.3389/fncel.2023.1105247
6. Nandhu MS, Paul J, Kuruvila KP, Abraham PM, Antony S, Paulose CS. Glutamate and NMDA receptors activation leads to cerebellar dysfunction and impaired motor coordination in unilateral 6-hydroxydopamine lesioned Parkinsons' rat: Functional recovery with bone marrow cells, serotonin and GABA. Mol Cell Biochem. 2011;353(1-2):47–57. https://doi.org/10.1007/s11010-011-0773-x
7. Vashisth MK, Hu J, Liu M, Basha SH, Yu C, Huang W. In-Silico discovery of 17alpha-hydroxywithanolide-D as a potential neuroprotective allosteric modulator of NMDA receptor targeting Alzheimer's disease. Sci Rep. 2024;14(1). https://doi.org/10.1038/srep42717
8. Re R, Pellegrini N, Proteggente A, Pannala A, Yang M, Rice-Evans C. Antioxidant activity applying an improved ABTS radical cation decolourisation assay. Free Radic Biol Med. 1999;26:1231–37. https://doi.org/10.1016/S0891-5849(98)00315-3
9. Mirke N, Shelke P, Malavdkar P, Jagtap P. In vitro protein denaturation inhibition assay of *Eucalyptus globulus* and *Glycine max* for potential anti-inflammatory activity. Innovations in Pharmaceuticals and Pharmacotherapy. 2020;8(2):28–31.
10. Daina A, Michielin O, Zoete V. SwissADME: A free web tool to evaluate pharmacokinetics, drug-likeness and medicinal chemistry friendliness of small molecules. Sci Rep. 2017;7(1):42717. https://doi.org/10.1038/srep42717
11. Sanner MF. Python: A Programming Language for Software Integration and Development. J. Mol. Graphics Model. 1999;17(1):57–61.
12. Ren X, Gelinas AD, Von Carlowitz I, Janjic N, Pyle AM. Structural basis for IL-1α recognition by a modified DNA aptamer that specifically inhibits IL-1α signaling. Nat Commun. 2017;8(1):810. https://doi.org/10.1038/s41467-017-00864-2
13. Lo SC, Li X, Henzl MT, Beamer LJ, Hannink M. Structure of the Keap1:Nrf2 interface provides mechanistic insight into Nrf2 signalling. EMBO Journal. 2006;25(15):3605–17. https://doi.org/10.1038/sj.emboj.7601243
14. Stroebel D, Buhl DL, Knafels JD, Chanda PK, Green M, Sciabola S, et al. A novel binding mode reveals two distinct classes of NMDA receptor GluN2B-selective antagonists. Mol Pharmacol. 2016;89(5):541–51. https://doi.org/10.1124/mol.115.103036
15. Wang C, Jiang Y, Ma J, Wu H, Wacker D, Katritch V, et al. Structural basis for molecular recognition at serotonin receptors. Science (1979). 2013;340(6132):610–14. https://doi.org/10.1126/science.1232807
16. Tiger M, Varnäs K, Okubo Y, Lundberg J. The 5-HT 1B receptor - a potential target for antidepressant treatment. Vol. 235, Psychopharmacology. Verlag: Springer; 2018. p. 1317–34. https://doi.org/10.1007/s00213-018-4872-1
17. Miller PS, Aricescu AR. Crystal structure of a human GABAA receptor. Nature. 2014;512(7514):270–75. https://doi.org/10.1038/nature13293
18. Trott O, Olson AJ. AutoDock Vina: Improving the speed and accuracy of docking with a new scoring function, efficient optimization and multithreading. J Comput Chem. 2010;31(2):455–61. https://doi.org/10.1002/jcc.21334
19. Banerjee P, Kemmler E, Dunkel M, Preissner R. ProTox 3.0: A web server for the prediction of toxicity of chemicals. Nucleic Acids Res. 2024;52(1):513–20. https://doi.org/10.1093/nar/gkae303
20. Bora NS, Bairy PS, Salam A, Kakoti BB. Antidiabetic and antiulcerative potential of *Garcinia lanceifolia* Roxb. bark. Futur J Pharm Sci. 2020;6(1):1–11. https://doi.org/10.1186/s43094-020-00101-6
21. Ghosh A, Banik S, Amin MN, Ahmed J. Evaluation of antinociceptive, antihyperglycemic and membrane stabilizing activities of *Garcinia lancifolia* Roxb. J Tradit Complement Med. 2018 ;8(2):303–07. https://doi.org/10.1016/j.jtcme.2017.04.009
22. Vasantharani S, Thirugnanasampandan R, Bhuvaneswari G. Qualitative and quantitative analysis of Precocene II, estimation of enzymatic, nonenzymatic antioxidant and cytotoxic potentials of methyl jasmonate-elicited shoot culture of *Ageratum conyzoides* Linn. J Appl Biol Biotechnol. 2023 Jan 1;11(1):93–99. https://doi.org/10.7324/JABB.2023.110114
23. Vieira C, Fetzer S, Sauer SK, Evangelista S, Averbeck B, Kress M, et al. Pro and anti-inflammatory actions of ricinoleic acid: Similarities and differences with capsaicin. Naunyn Schmiedebergs Arch Pharmacol. 2001;364(2):87–95. https://doi.org/10.1007/s002100100427
24. Mascolo N, Barbato F, Grumetto L, Quaglia F, Capasso F. Ricinoleic acid, the active ingredient of castor oil, increases Nitric Oxide Synthase activity in the rat ileum and colon. Pharm Biol. 1997;35(5):364–70.
25. Wang T, Lee SL, Da Silva JAC, Hammond E, Yao L. Determination of oxidation of methyl ricinoleates.Journal of the American Oil Chemists' Society. 2015;92(6):871–80. https://doi.org/10.1007/s11746-015-2649-9
26. Neganova M, Liu J, Aleksandrova Y, Vasilieva N, Semakov A, Yandulova E, et al. Development of Neuroprotective Agents for the Treatment of Alzheimer's Disease using Conjugates of Serotonin with Sesquiterpene Lactones. Curr Med Chem. 2024;31(5):529–51. https://doi.org/10.2174/0929867330666221125105253

27. Pairas GN, Tsoungas PG. H-Bond: The Chemistry-Biology H-Bridge. Vol. 1, Chemistry Select. 2016:1(15):4520–32. https://doi.org/10.1002/slct.201600770

28. Tóth G, Bowers SG, Truong AP, Probst G. The Role and Significance of Unconventional Hydrogen Bonds in Small Molecule Recognition by Biological Receptors of Pharmaceutical Relevance. Curr Pharm Des. 2007;13(34):3476–93. https://doi.org/10.2174/138161207782794284

29. Hunter CA. Aromatic Interactions in Proteins, DNA and Synthetic Receptors. Philos Trans R Soc Lond A Phys Eng Sci. 1993;345 (1674):77–85. https://doi.org/10.1098/rsta.1993.0119

30. Mierke DF, Pellegrini M. Receptor-Ligand Interactions. Encyclopedia of Endocrine Diseases. 2004;163–66. https://doi.org/10.1016/B0-12-475570-4/01127-6

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