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Molecular Docking Analysis of Phytoconstituents as Potential EGFR Inhibitors: Insights into Natural Scaffolds for Anticancer Drug Discovery

(Running Title: Molecular Docking of Natural EGFR Scaffolds)

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ABSTRACT

Background: The epidermal growth factor receptor (EGFR) is a cell surface receptor that regulates cell proliferation and survival; its abnormal activation, which often occurs in non-small-cell lung cancer (NSCLC), causes tumor progression and resistance to tyrosine kinase inhibitors (TKIs). Although next-generation TKIs have shown some improvements over previous treatments, there are still major challenges with resistance and toxicity. Natural phytoconstituents represent a potential reservoir of structurally diverse scaffolds for safer anticancer drug discovery. **Objective:** This study aimed to assess the binding ability of selected phytoconstituents against the EGFR kinase domain (PDB ID: 6JXT) by molecular docking. Methods: Seven phytoconstituents, including arjunolic acid, gallic acid, kaempferol, lupeol, quercetin, quisqualic acid, and rutin, were retrieved from PubChem and prepared using Open Babel (PyRx). The EGFR structure (PDB ID: 6JXT) was processed and docked with ligands using AutoDock Vina in PyRx 0.8. Results of docking were ranked by binding affinities (docking scores, kcal/mol), and receptor–ligand interactions were analyzed using BIOVIA Discovery Studio Visualizer**. Results:** Docking scores ranged from –5.6 to –9.7 kcal/mol. Arjunolic acid (–9.7) and quercetin (–9.6) had the highest affinities, with multiple stabilizing interactions, while lupeol (–8.6), rutin (–8.3), gallic acid, kaempferol, and quisqualic acid showed weaker interactions (–5.6). **Conclusion:** Arjunolic acid and quercetin appeared to be the most promising natural scaffolds for EGFR inhibition, with lupeol and rutin as secondary candidates, but further validation through molecular dynamics, ADMET

profiling, and biological assays is needed to confirm their therapeutic potential in EGFR-targeted cancer therapy.

Keywords: EGFR, molecular docking, phytoconstituents, arjunolic acid, quercetin, flavonoids, triterpenoids, NSCLC.

INTRODUCTION

The Epidermal Growth Factor Receptor (EGFR), a transmembrane tyrosine kinase, is involved in the regulation of basic cellular processes such as proliferation, differentiation, and survival. Dysregulated EGFR signalling, often due to overexpression or activation mutations, is strongly involved in oncogenesis, particularly in non-small cell lung cancer (NSCLC), head and neck squamous cell carcinoma (HNSCC), breast cancer, and glioblastoma [1-5]. Mutations such as L858R, exon 19 deletions, and the gatekeeper T790M substitution drive tumor progression and therapeutic resistance, highlighting EGFR as a critical drug target [6-9].

Although several generations of EGFR tyrosine kinase inhibitors (TKIs) such as gefitinib, erlotinib, afatinib, osimertinib, and investigational compounds like sunvozertinib and asandeutertinib have demonstrated clinical utility, limitations persist due to acquired resistance, off-target toxicities, and tumor heterogeneity [10-14]. Consequently, attention has shifted toward the discovery of novel, safe, and selective EGFR modulators.

Natural products, particularly phytoconstituents such as flavonoids, triterpenoids, and phenolic acids, are promising sources of lead compounds owing to their structural diversity, relatively low toxicity, and multi-targeted bioactivity [15-19]. Quercetin, kaempferol, rutin, lupeol, and arjunolic acid are notable examples with reported anticancer potential through inhibition of EGFR and downstream pathways, including PI3K/AKT and MAPK signaling [20-24]. Recent computational investigations combining molecular docking, molecular dynamics (MD), and density functional theory (DFT) analysis have identified phytochemicals from Moringa oleifera, Mangifera indica, and other plants as effective EGFR modulators [25-29]. Moreover, phytoconstituents can act synergistically with synthetic TKIs or serve as templates for novel scaffold development [30-32].

Computational tools such as molecular docking, MD simulations, and ADMET profiling provide rapid and cost-effective strategies to screen natural compounds and predict their inhibitory potential against EGFR [33-36]. Such in silico methods are increasingly

recognized as essential in early-stage drug discovery, bridging the gap between traditional herbal pharmacology and modern targeted therapy [37-40].

This study assessed the binding affinity of seven phytoconstituted substances (arjunolic acid, gallic acid, kaempferol, lupeol, quercetin, quisqualic acid, and rutin) to the EGFR kinase domain (PDB ID 6JXT). This work aims to identify natural scaffolds with the potential for further optimization into new EGFR inhibitors through molecular docking and interaction analysis.

MATERIALS AND METHODS

Selection and Preparation of Target Protein

The crystal structure of the target protein (PDB ID: 6JXT) was retrieved from the RCSB Protein Data Bank and prepared for docking using BIOVIA Discovery Studio Visualizer by removing co-crystallized ligands, water molecules, and heteroatoms, adding hydrogen atoms to correct valency, and identifying and selecting the active site based on literature reports and co-crystal references.

Ligand Selection and Preparation

Seven phytoconstituents (Arjunolic acid, Gallic acid, Kaempferol, Lupeol, Quercetin, Quisqualic acid, and Rutin) were selected based on reported biological activities, and their structures were retrieved from the PubChem database in SDF or Mol2 formats. Ligand preparation was performed in Open Babel (PyRx), involving energy minimization with the MMFF94 force field and conversion to PDBQT format for docking.

Molecular Docking Protocol

Molecular docking was carried out using AutoDock Vina integrated in PyRx 0.8. The prepared receptor (6JXT) and ligands were imported, and a grid box was defined around the active site to encompass all potential binding regions. Each ligand was docked independently with default Vina parameters and an exhaustiveness value of 8. Docking results were ranked by binding affinity (kcal/mol), with more negative values indicating stronger predicted interactions.

Visualization and Analysis

Post-docking analysis was conducted in BIOVIA Discovery Studio Visualizer to examine ligand–receptor interactions, including hydrogen bonding, hydrophobic contacts, and π–π stacking, and to evaluate ligand orientation within the binding site.

Data Interpretation

Docking scores were comparatively analyzed to evaluate the binding potential of each phytoconstituent, with Quisqualic acid showing relatively weak binding affinity, indicative of poor interaction with the 6JXT receptor [41-43].

RESULTS AND DISCUSSION

Docking Result

The molecular docking results of the selected phytoconstituents with the 6JXT receptor are summarized in Table 1. Docking scores ranged from –5.6 to –9.7 kcal/mol, indicating differential binding potentials among the tested compounds.

Table 1: Docking scores (kcal/mol) of phytoconstituents with receptor **6JXT**, showing the strongest affinities for Arjunolic acid (-9.7) and Quercetin (-9.6).

S. No.	Receptor	Phytoconstituents	Class	Dock Score
1.	6JXT	Arjunolic Acid	Pentacyclic triterpenoid	-9.7
2.	6JXT	Gallic Acid	Phenolic compound	-5.6
3.	6JXT	Kaempferol	Flavonoid	-5.6
4.	6JXT	Lupeol	Triterpenoid	-8.6
5.	6JXT	Quercetin	Flavonoid	-9.6
6.	6JXT	Quisqualic acid	Flavonoid	-5.6
7.	6JXT	Rutin	Flavonoid	-8.3

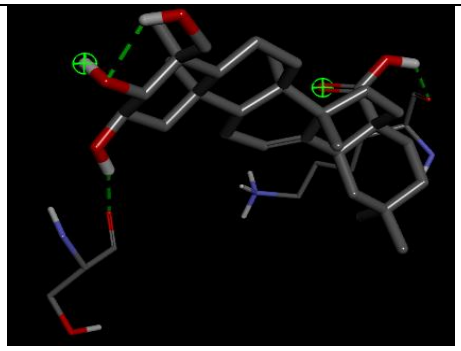
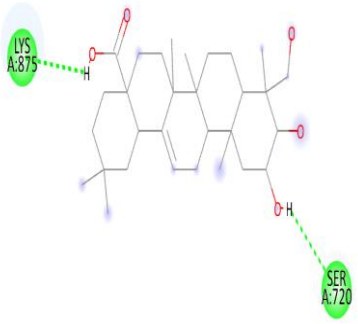
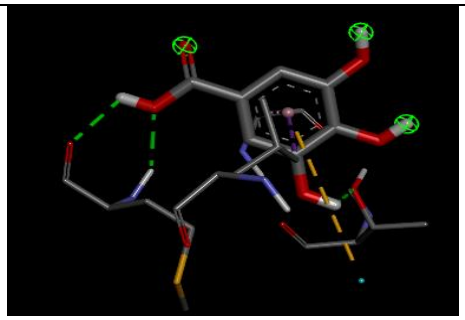
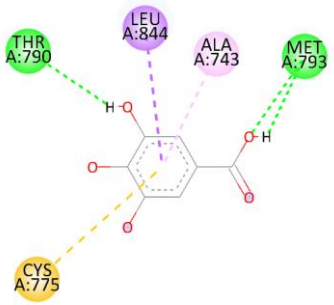
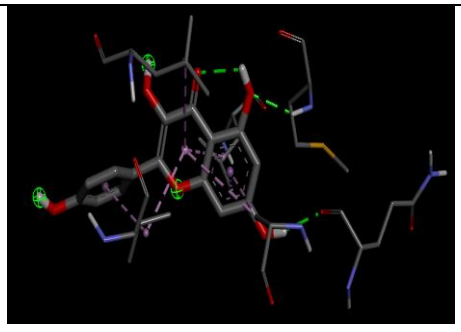
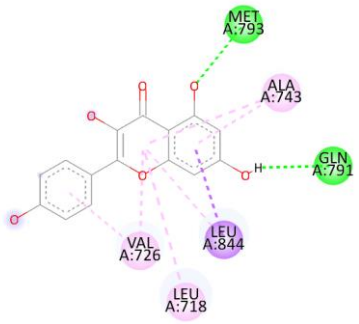
The molecular docking analysis of seven phytoconstituents with the 6JXT receptor—the epidermal growth factor receptor (EGFR; residues 696–1022 wild-type) in complex with the inhibitor AZD9291 (Osimertinib)—revealed a broad range of binding affinities, spanning from –5.6 to –9.7 kcal/mol. Among these, arjunolic acid (–9.7 kcal/mol) and quercetin (–9.6 kcal/mol) exhibited the strongest binding, suggesting a high potential for stable and robust interactions with the EGFR active site. Lupeol (–8.6 kcal/mol) and rutin (–8.3 kcal/mol) also demonstrated appreciable binding affinities, albeit slightly lower than the top-scoring ligands. In contrast, gallic acid, kaempferol, and quisqualic acid each displayed weaker binding scores (–5.6 kcal/mol), indicating suboptimal interactions and potentially limited stability within the EGFR binding pocket (Table 2).

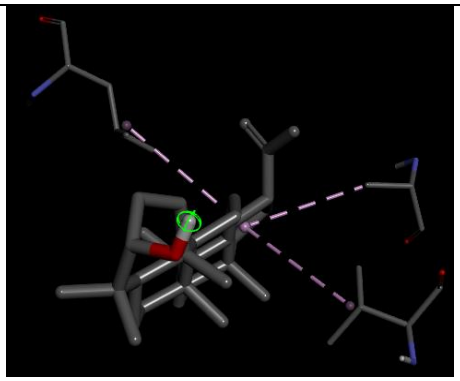
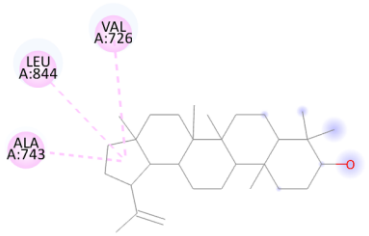
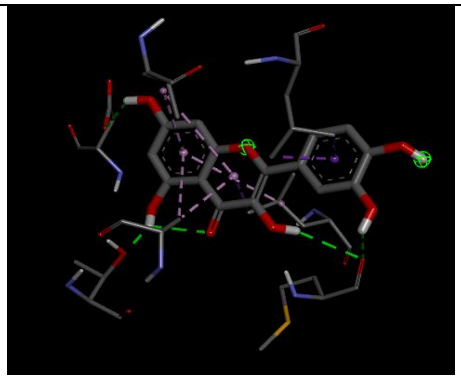
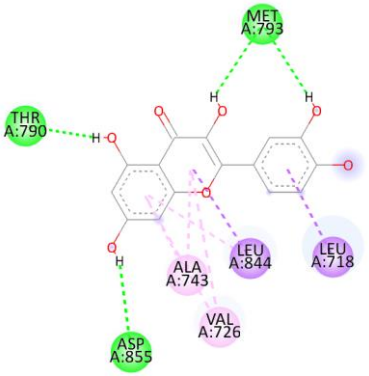
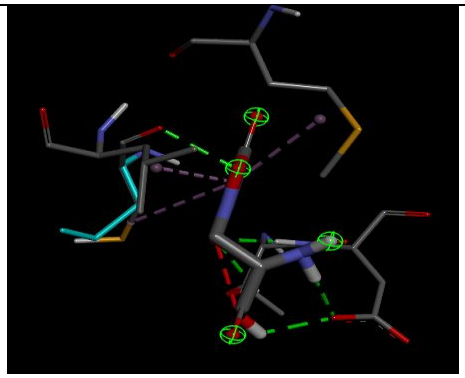
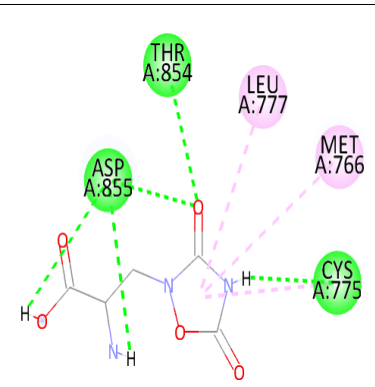
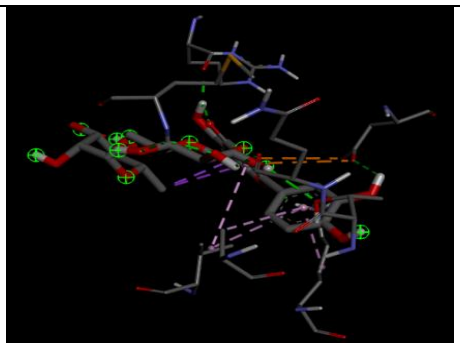
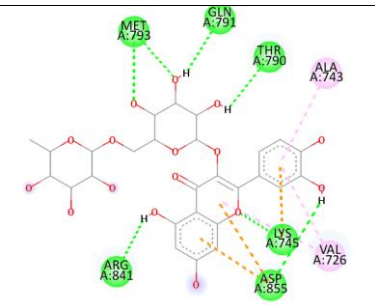
Post-docking analysis revealed that arjunolic acid and quercetin engage EGFR via multiple **hydrogen bonds, hydrophobic contacts, and π–π stacking interactions**, which likely account for their superior docking profiles. Arjunolic acid's pentacyclic triterpenoid core may establish numerous van der Waals and hydrophobic contacts, while its hydroxyl groups

facilitate hydrogen bonding [24]. Quercetin's flavonoid structure, with its polyhydroxylated aromatic rings, supports both hydrogen bonding and π–π-electron interactions [44]. Its strong docking score in this study corroborates earlier reports of its therapeutic potential.

Lupeol's moderate affinity aligns with its known anti-inflammatory and anticancer mechanisms, often involving receptor-mediated pathways [22]. Rutin's enhanced binding is presumably due to its glycosidic substituents, which offer additional hydrogen-bonding capacity within the EGFR binding pocket [21].

Table 2: Ligand–receptor interactions and 2D structures of selected phytoconstituents docked with 6JXT

Phytoconstituents	Ligand interaction	2d structure
Arjunolic Acid		
Gallic Acid		
Kaempferol		

Lupeol		
Quercetin		
Quisqualic acid		
Rutin		

The weaker interactions seen with gallic acid and kaempferol likely stem from their smaller molecular sizes and reduced binding complementarity, and quisqualic acid's poor affinity suggests unfavorable orientation or limited stabilizing contacts.

Mechanistic Implications Linked to EGFR Function

EGFR is a receptor tyrosine kinase that is central to the regulation of cell proliferation, differentiation, and survival, mediated by ligand-induced dimerisation and subsequent autophosphorylation. Dysregulated EGFR signaling is involved in many cancers, particularly non-small cell lung cancer (NSCLC), with drug resistance associated with T790M. The strong binding of arjunolic acid and quercetin to the EGFR kinase domain indicates that they may act as new modulators or inhibitors of EGFR activity, possibly blocking the ATP binding or stabilising the inactive conformations. Such binding could attenuate downstream oncogenic signaling pathways like PI3K/AKT or MAPK, aligning with their reported bioactivities. Indeed, flavonoids like quercetin are known to interfere with EGFR-mediated signaling, while triterpenoids such as arjunolic acid are recognized for targeting signaling proteins in cancer pathways.

Taken together, these findings highlight **arjunolic acid** and **quercetin** as lead scaffolds for further development into EGFR modulators. **Lupeol** and **rutin** also warrant further biochemical and biophysical evaluation. The weaker-scoring compounds may still hold value as structural templates for future optimization.

CONCLUSION

This study employed molecular docking to assess the inhibitory potential of seven phytoconstituents—arjunolic acid, gallic acid, kaempferol, lupeol, quercetin, quisqualic acid, and rutin—against the EGFR kinase domain (PDB ID: 6JXT). Among these, arjunolic acid and quercetin exhibited the strongest binding affinities, characterized by multiple hydrogen bonds, hydrophobic interactions, and π–π stacking, suggesting robust interaction within the EGFR active site. Lupeol and rutin demonstrated moderate binding, while gallic acid, kaempferol, and quisqualic acid showed relatively weaker affinities, indicating suboptimal receptor complementarity.

These findings are consistent with prior computational and experimental studies reporting the potent anti-EGFR activity of triterpenoids and flavonoids [6, 13, 20, 22, 23]. For example, quercetin has been extensively validated as an EGFR modulator through both docking and in vitro studies [20, 26, 40], while arjunolic acid and related triterpenoids are increasingly recognized as multi-target anticancer scaffolds [23, 24, 29]. Lupeol and rutin have also shown promising activity against EGFR-driven signaling pathways, reinforcing their potential for further optimization [21, 22].

What is important is that EGFR dysregulation remains a major driver of NSCLC and other cancers, and despite progress with new generation TKIs such as osimertinib and sunvaxertinib, acquired resistance remains a major challenge [10-14, 37]. Natural phytoconstituents, with their favorable safety profiles and multi-targeted mechanisms, could complement or inspire novel EGFR inhibitors that overcome resistance [6, 18, 30, 45].

While molecular docking provides rapid and cost-effective insights, it remains predictive in nature. Further validation through molecular dynamics simulations, ADMET profiling, biochemical kinase inhibition assays, and in vivo efficacy studies will be crucial to confirm the therapeutic relevance of these compounds [39, 46, 47]. Future work may also explore synergistic interactions of phytochemicals with approved TKIs to enhance efficacy and mitigate resistance [30, 32].

In conclusion, this study highlighted arjunolic acid and quercetin as promising natural scaffolds for EGFR-targeted cancer therapeutics, with lupeol and rutin as minor candidates. By combining computational screening with phytochemical drug discovery, this study adds to growing evidence that plant compounds may play a key role in the development of next-generation EGFR inhibitors.

CONFLICT OF INTEREST

The authors have no conflicts of interest regarding this investigation**.**

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REFERENCES

[1] Halder S, Basu S, Lall SP, Ganti AK, Batra SK, Seshacharyulu PJEoott. Targeting the EGFR signaling pathway in cancer therapy: What's new in 2023? 2023;27(4-5):305-24.

[2] Zhang YJPR. Targeting epidermal growth factor receptor for cancer treatment: abolishing both kinase-dependent and kinase-independent functions of the receptor. 2023;75(6):1218-32.

[3] Li S, Sun Y. Phytochemicals targeting epidermal growth factor receptor (EGFR) for the prevention and treatment of HNSCC: A review. Medicine. 2023;102(40):e34439.

[4] Yang H, Zhang Z, Liu Q, Yu J, Liu C, Lu WJM. Identification of dual-target inhibitors for epidermal growth factor receptor and AKT: virtual screening based on structure and molecular dynamics study. 2023;28(22):7607.

[5] Khan MN, Farooq U, Khushal A, Wani TA, Zargar S, Khan SJPO. Unraveling potential EGFR kinase inhibitors: Computational screening, molecular dynamics insights, and MMPBSA analysis for targeted cancer therapy development. 2025;20(5):e0321500.

[6] Sepay N, Mondal R, Al-Muhanna MK, Saha DJNJoC. Identification of natural flavonoids as novel EGFR inhibitors using DFT, molecular docking, and molecular dynamics. 2022;46(20):9735-44.

[7] Saeed ME, Yücer R, Dawood M, Hegazy M-EF, Drif A, Ooko E, et al. In silico and in vitro screening of 50 curcumin compounds as EGFR and NF-κB inhibitors. 2022;23(7):3966.

[8] Noor MAA, Haq MM, Chowdhury MAR, Tayara H, Shim H, Chong KTJP. In silico exploration of novel EGFR kinase mutant-selective inhibitors using a hybrid computational approach. 2024;17(9):1107.

[9] Wang S, Xu X, Pan C, Guo Q, Li Q, Wan S, et al. Identification of new EGFR inhibitors by structure-based virtual screening and biological evaluation. 2024;25(3):1887.

[10] Yuan C, Yu J-Y, Zeng C-X, Wang M-C, Zhang S, Huang Y-B, et al. New advances in the treatment of EGFR exon20ins mutant advanced NSCLC. 2025;15(4):1852.

[11] Han B, Liu J, Wu L, Zhao Y, Zhang W, Chen B, et al. Safety, pharmacokinetics, and efficacy of asandeutertinib in advanced EGFR-mutated NSCLC: a phase 1 dose-escalation and dose-expansion study. 2025.

[12] Tortora G, Ciardiello F, Gasparini GJNcpO. Combined targeting of EGFR-dependent and VEGF-dependent pathways: rationale, preclinical studies and clinical applications. 2008;5(9):521-30.

[13] Al-Wahaibi LH, Elshamsy AM, Ali TF, Youssif BG, Bräse S, Abdel-Aziz M, et al. Design and synthesis of new dihydropyrimidine derivatives with a cytotoxic effect as dual EGFR/VEGFR-2 inhibitors. 2024;9(32):34358-69.

[14] Al Mashud MA, Kumer A, Jahan I, Somrat MMH, Talukder MEK, Rahman MM, et al. Chemoinformatics analysis of Mangifera indica leaves extracted phytochemicals as potential EGFR kinase modulators. 2025;13:1524384.

[15] Serag MI, Tawfik SS, Eisa HM, Badr SMJDDR. Design, Synthesis, Biological Evaluation and Molecular Docking Study of New 1, 3, 4-Thiadiazole-Based Compounds as EGFR Inhibitors. 2025;86(1):e70035.

[16] Ahmmed S, Bhuia MS, Chowdhury R, Munshi MH, Eity TA, Kamli H, et al. Potential Therapeutic Efficacy of Ferulic Acid and Its Derivatives in the Management of Cancers: A Comprehensive Analysis With Mechanistic Insight. 2025;2025(1):2256871.

[17] Sangande F, Julianti E, Tjahjono DH. Ligand-Based Pharmacophore Modeling, Molecular Docking, and Molecular Dynamic Studies of Dual Tyrosine Kinase Inhibitor of EGFR and VEGFR2. 2020;21(20):7779.

[18] Rezaee P, Rezaee S, Maaza M, Arab SSJCiB, Medicine. Screening of BindingDB database ligands against EGFR, HER2, Estrogen, Progesterone and NF-κB receptors based on machine learning and molecular docking. 2024;183:109279.

[19] Biswas P, Mathur D, Dinesh J, Dinesh HK, Desai BMA, editors. Computational analysis using multi-ligand simultaneous docking of withaferin A and garcinol reveals enhanced BCL-2 and AKT-1 inhibition. 2025 International Conference on Innovation in Computing and Engineering (ICE); 2025: IEEE.

[20] Chen R, Hang L, Wang FJapa. Mechanism of Quercetin in Inhibiting Triple-Negative Breast Cancer by Regulating T Cell-Related Targets: An Analysis Based on Single-Cell Sequencing and Network Pharmacology. 2025.

[21] Ganeshpurkar A, Saluja AKJSpj. The pharmacological potential of rutin. 2017;25(2):149-64.

[22] Saleem MJCl. Lupeol, a novel anti-inflammatory and anti-cancer dietary triterpene. 2009;285(2):109-15.

[23] Nanda A, H Ansari S, Khatkar SJCOC. Arjunolic acid: a promising antioxidant moiety with diverse biological applications. 2017;21(4):287-93.

[24] Hussain SA, Panjagari NR, Singh R, Patil GJCRiFS, Nutrition. Potential herbs and herbal nutraceuticals: food applications and their interactions with food components. 2015;55(1):94-122.

[25] Maleki MSM, Sardari S, Alavijeh AG, Madanchi HJIjopr, therapeutics. Recent patents and FDA-approved drugs based on antiviral peptides and other peptide-related antivirals. 2022;29(1):5.

[26] Aljabali AA, Obeid MA, Bashatwah RM, Qnais E, Gammoh O, Alqudah A, et al. Phytochemicals in cancer therapy: a structured review of mechanisms, challenges, and progress in personalized treatment. 2025:e202402479.

[27] Suwal N, Thapa R, Bashyal S, Sugandhi VV, Subedi S, Panth N, et al. Targeting oncogenic signaling pathways in lung Cancer: The emerging role of nobiletin, a flavonoid from citrus peel. Food Bioscience. 2025;69:106887.

[28] Zamanian MY, Shahbazi T, Kazmi SW, Hussien BM, Sharma A, Qasim MT, et al. Effects of resveratrol on nonmelanoma skin Cancer (NMSC): A Comprehensive review. 2024;12(11):8825-45.

[29] Lephatsi MM, Choene MS, Kappo AP, Madala NE, Tugizimana FJM. An integrated molecular networking and docking approach to characterize the metabolome of Helichrysum splendidum and its pharmaceutical potentials. 2023;13(10):1104.

[30] Varne AA, Bhatia MS, Ashtek SSJPCJ. A systematic review on chemical actives from plant sources, targets and chemotherapy for triple-negative breast cancer. 2023;57(5):694-702.

[31] Sepay N, Mondal R, Al-Muhanna M, Saha D. Identification of natural flavonoids as novel EGFR inhibitors using DFT, molecular docking, and molecular dynamics. New Journal of Chemistry. 2022.

[32] Tiwari P, Park K-IJAS. Seed Biotechnologies in Practicing Sustainable Agriculture: Insights and Achievements in the Decade 2014–2024. 2024;14(24):11620.

[33] Li K, Duan M, Lu Q, Liu J, He M, Zhang YJi. Advances in Neuroscientific Mechanisms and Therapies for Glioblastoma. 2025.

[34] Gupta S, Kumar A, Tejavath KKJFJoPS. A pharmacognostic approach for mitigating pancreatic cancer: emphasis on herbal extracts and phytoconstituents. 2021;7(1):96.

[35] Yassen A, Nawareg N, Husseiny E, Elsayed M, Elshihawy H. 1,2,3-Triazole-Based Hybrids as EGFR Inhibitors: An overview. Records of Pharmaceutical and Biomedical Sciences. 2025;9:84-91.

[36] Saponara S, Carosati E, Mugnai P, Sgaragli G, Fusi F. The flavonoid scaffold as a template for the design of modulators of the vascular Ca(v) 1.2 channels. British journal of pharmacology. 2011;164(6):1684-97.

[37] Zhai D, Zhang Q, Lu X, You Q, Wang LJTI. Global first-in-class drugs approved in 2023–2024: Breakthroughs and insights. 2025;6(4).

[38] Lee HYJ, Meng M, Liu Y, Su T, Kwan HY. Medicinal herbs and bioactive compounds overcome the drug resistance to epidermal growth factor receptor inhibitors in non-small cell lung cancer. Oncology letters. 2021;22(3):646.

[39] Huang L, Dong Y, Li J, Yang X, Li X, Wu J, et al. Epidermal Growth Factor Receptor (EGFR)-Targeting Peptides and Their Applications in Tumor Imaging Probe Construction: Current Advances and Future Perspectives. 2025;14(8):1011.

[40] Simão AL, Santos SS, Moreira JAF, Silva F, Oliveira R, Roxo C, et al. Depression- and exercise-associated stimuli exert contrasting effects on neural stem cell activity and paracrine signaling. 2025;1871(8):167997.

[41] K. Lim S, Othman R, Yusof R, H. Heh CJCC-ADD. Rational Drug Discovery of HCV Helicase Inhibitor: Improved Docking Accuracy with Multiple Seeding in AutoDock Vina and In Si tu Minimization. 2017;13(2):160-9.

[42] Ayodele PF, Bamigbade A, Bamigbade OO, Adeniyi IA, Tachin ES, Seweje AJ, et al. Illustrated procedure to perform molecular docking using PyRx and Biovia discovery studio visualizer: a case study of 10kt with atropine. 2023;6(1).

[43] Kashid S, Suttee A, Kadam P, editors. In Silico Prediction and Screening of Potential Immunomodulators Using Autodock Vina. Techno-Societal 2016, International Conference on Advanced Technologies for Societal Applications; 2022: Springer.

[44] Nanda A, Ansari S, Khatkar S. Arjunolic Acid: A Promising Antioxidant Moiety with Diverse Biological Applications. Current Organic Chemistry. 2017;21:287-93.

[45] Lee HYJ, Meng M, Liu Y, Su T, Kwan HYJOL. Medicinal herbs and bioactive compounds overcome the drug resistance to epidermal growth factor receptor inhibitors in non-small cell lung cancer. 2021;22(3):646.

[46] Mousavi Maleki MS, Sardari S, Ghandehari Alavijeh A, Madanchi HJIJoPR, Therapeutics. Recent patents and FDA-approved drugs based on antiviral peptides and other peptide-related antivirals. 2022;29(1):5.

[47] Suwal N, Thapa R, Bashyal S, Sugandhi VV, Subedi S, Panth N, et al. Targeting oncogenic signaling pathways in lung Cancer: The emerging role of nobiletin, a flavonoid from citrus peel. 2025;69:106887.

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