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Research Article

Computational Docking Analysis for Identifying Natural Inhibitors of TEM-1 β-Lactamase

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ABSTRACT

TEM-1 β-lactamase is a class A enzyme widely distributed among pathogenic bacteria and plays a major role in the hydrolysis of β-lactam antibiotics, contributing to antibiotic resistance. The present study investigated the inhibitory potential of a flavonoid-derived phytocompound against this enzyme using computational molecular docking analysis. The interaction between TEM-1 β-lactamase and Eupatin -flavonoid derived compound was evaluated and compared with the reference inhibitor Clavulanic acid. The reference inhibitor showed binding energies ranging from −4.7 to −6.5 kcal/mol, while the phytocompound demonstrated a stronger binding affinity with a docking score of approximately −7.35 kcal/mol. The ligand was found to occupy the catalytic pocket of the enzyme and formed stable interactions with key active-site residues, indicating its potential to inhibit TEM-1 β-lactamase activity. Overall, the results suggest that this phytocompound may serve as a promising lead candidate for the development of new β-lactamase inhibitors. However, additional studies such as molecular dynamics simulations, pharmacokinetic analysis, and experimental validation are required to further confirm its stability and therapeutic potential in combating β-lactamase-mediated antibiotic resistance.

1. Introduction

Antibiotic resistance is a major global health issue in which bacteria become able to multiply and survive even when exposed to the drugs which made for kill them or stop their growth. This happens through various natural processes, such as genetic mutations and the production of enzymes that break down antibiotics, including penicillin and cephalosporins. Some bacteria are capable of forming biofilms, which enhance protection against various antibiotics treatments and leads to increased antimicrobial resistance (Sharma et al., 2025). Bacteria can also gain resistant genes against antibiotics from other microbes through the transfer of genetic material, allowing resistance to spread quickly across different species(Binek et al., 2019). Recent statistics India has seen a notable increase in drug-resistant infections (Bhatia, 2024). Common examples include Methicillin-resistant Staphylococcus aureus (MRSA) and Vancomycin-resistant Enterococci (VRE), which are resistant to all β-lactam antibiotics and difficult to treat (Nandhini et al., 2022),(Tacconelli & Cataldo, 2008). Multi-drug-resistant bacteria, such as Mycobacterium tuberculosis (MDR-TB), are also major concerns. Additionally, Gram-negative bacteria like Escherichia coli (E. coli), Klebsiella pneumoniae, and Pseudomonas aeruginosa have acquired resistance to multiple antibiotics (Vyas, 2012; Kumar et al., 2020; Porika et al., 2014).

The rising prevalence of antibiotic resistance places a heavy strain on the healthcare system, resulting in an increase in healthcare-associated infections, which ultimately leads to longer hospital stays, higher treatment costs, and increased patient vulnerability, raising the risk of complications and death (Singh, 2023; Swapna et al., 2024: Lunavath et al., 2013; Ramana & Chaudhury, 2012). Many infections now require stronger, more expensive treatment and medicines that may have severe side effects. As a result, routine medical procedures such as surgeries and cancer treatments have become riskier due to the threat of untreatable infections. Several factors contribute to the rise of antibiotic resistance in India, including the overuse and misuse of antibiotics, self-medication, incomplete treatment courses, and poor infection control practices (Rani, 2024; Sachdev et al., 2022; Namthabad et al., 2014). Limited awareness and poor hygiene and sanitation in some areas further accelerate the spread of resistant bacteria. One important factor in antibiotic resistance is, the enzyme TEM-1 β-lactamase found mainly in Gram-negative bacteria like Escherichia coli, this enzyme breaks down β-lactam antibiotics by destroying their active structure, making the

drugs ineffective (Tooke et al., 2019) (Cantón, 2014). Since the genes for this enzyme can easily spread between bacteria, TEM-1 plays a key role in the rapid increase of resistance, making infections harder to treat.

Molecular docking is a computational technique used to predict how ligands bind to a target protein and determine its bindng strength. It plays an main role in drug discovery by helping researchers to understand the Receptor-ligand interactions and identify potential drug candidates (Trott & Olson, 2010). Along with molecular dynamics (MD) simulations, it provides valuable insights into binding mechanisms, stability, and the energetics of interactions. These methods also help estimate important parameters such as binding energy and inhibition constant (Ki), which are essential for evaluating the effectiveness of a compound (Meng et al., 2011). Molecular docking is widely used to optimise the lead compounds by improving their binding affinity and specificity. Overall, it is a key computer aided drug design approach based on mathematical algorithms that predict how well a drug molecule can interact with its biological target, making it a valuable tool in pharmaceutical research.

2. Materials and Methods

2.1. Computational Tools and Databases Used for In Silico Analysis

The computational study was performed using multiple software tools. AutoDockTools 1.5.7 was used for molecular docking analysis, while Discovery Studio 2025 – Biovia was employed for protein and ligand optimization and for 2D and 3D visualization. UCSF Chimera 2025 was utilized for active site identification and surface structure analysis(Pettersen et al., 2004). Phytochemical data were obtained from the IMPPAT database and Pubchem database, and Open Babel was used to convert file formats compatible with AutoDock. Additionally, SwissADME, pkCSM, and ProTox-II web servers were used to predict ADMET properties. All tools were operated on a personal computer.

2.2. Selection of Target Protein

The target enzyme, TEM-1 β-lactamase (PDB ID: 1ERM)(Ness et al., 2000), was selected from the RCSB Protein Data Bank (PDB) https://www.rcsb.org/structure/1ERM , due to its well-established role in bacterial antibiotic resistance. TEM-1 β-lactamase, derived from Escherichia coli, belongs to class A β-lactamases and is responsible for hydrolyzing β-lactam antibiotics such as penicillin and ampicillin, thereby rendering them ineffective. The three-dimensional crystal structure of TEM-1 β-lactamase (1ERM) was retrieved in PDB format, which provides high-resolution structural details suitable for docking studies (Figure-1). The structure consists of a single polypeptide chain with well-defined active site residues, including Ser70, Lys73, Ser130, and Glu166, which are crucial for catalytic activity. Prior to docking analysis,

2.3. Protein Preparation for Docking Simulation

The retrieved protein structure of TEM-1 β-lactamase (PDB ID: 1ERM) was prepared for molecular docking using AutoDock Tools (ADT) v1.5.6 and Discovery Studio Visualizer (Biovia) to ensure accuracy and reliability of the docking results. The initial PDB file was processed by removing all water molecules, co-crystallized ligands, and other heteroatoms that could interfere with ligand binding. The cleaned protein structure was then subjected to energy minimization using the Steepest Descent algorithm in ADT to eliminate steric clashes and improve structural stability. Subsequently, Kollman charges were assigned, and Gasteiger charges were added to the protein to facilitate proper electrostatic interaction calculations. Non-polar hydrogens were merged, while polar hydrogens were retained to maintain hydrogen bonding interactions within the active site. Finally, the prepared and refined protein structure was saved in PDBQT format, making it suitable for further docking simulations

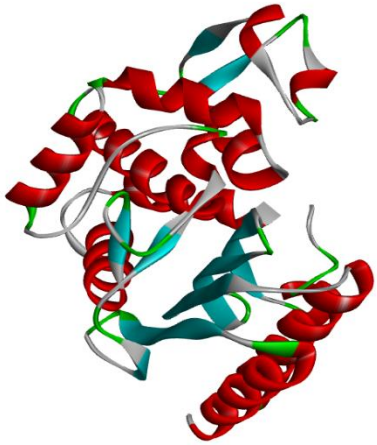

Fig 1. Visualization of 3D structure of TEM -1 β Lactamases (PDB ID: 1ERM) retrieved from RCSB database

2.4. Ligand Selection and Preparation

Potential inhibitors were selected from the IMPATT and PUBCHEM database focusing on flavonoid derived phytocompound with drug-like properties. Selection was based on Lipinski's rule of five, considering molecular weight, hydrogen bond donors and acceptors, and lipophilicity for oral bioavailability(Lipinski et al., 2001). Six flavonoid derivatives were chosen as lead compounds for docking studies. Their three-dimensional structures were obtained in SDF format and converted to PDB format using Open Babel v3.1.1. The ligands were then prepared by adding Gasteiger charges and defining rotatable bonds in AutoDock Tools (ADT) v1.5.6, and the final structures were saved in PDBQT format for docking with TEM-1 β-lactamase.

2.5. Molecular Docking simulations studies

Molecular docking studies were carried out using AutoDock 4.2 and AutoDock Tools (ADT) v1.5.6. The target, TEM-1 β-lactamase, was prepared in PDBQT format by adding Kollman and Gasteiger charges. Docking simulations employed the Lamarckian Genetic Algorithm (LGA) with parameters set to ensure extensive conformational sampling: 150 independent runs, 50 million energy evaluations, a maximum of 30,000 generations, a population size of 200, mutation rate of 0.02, crossover rate of 0.8, and an elitism value of 1. The docking grid was centred on the ATP-binding site (x = 34.014, y = 32.031, z = 28.015) with dimensions of 90 × 90 × 90 points and a spacing of 0.375 Å, encompassing the entire binding pocket. Protein-ligand complexes were assessed based on binding free energy (ΔG), inhibition constant (Ki), and key molecular interactions, including hydrogen bonds, hydrophobic contacts, and π-stacking. Visualization and detailed analysis were performed using Discovery Studio Visualizer, enabling identification of the most stable and biologically relevant binding

conformations, which were further considered as potential TEM-1 β-lactamase inhibitors.

2.6. ADMET Prediction

The ADMET properties of the selected compounds were predicted using established in silico tools, including SwissADME, pkCSM, and ProTox-II. Physicochemical properties such as lipophilicity (logP) and bioavailability were analyzed using SwissADME(Daina et al., 2017). While pharmacokinetic parameters including absorption (HIA), distribution (VDs), metabolism (CYP3A4 inhibition), and excretion (total clearance) were evaluated through pkCSM(Pires et al., 2015). Toxicity aspects, such as LD50 values and toxicity classification, were determined using ProTox-II(Banerjee et al., 2024). These computational methods were applied to comprehensively assess the pharmacokinetic characteristics and safety profiles of the compounds before further investigation.

3. Results and Discussion

3.1. Binding Affinity and Energy Profiles

The binding energy (ΔG, kcal/mol) analysis demonstrated notable differences in the interactions between the ligands and TEM-1 β-lactamase. Among the compounds tested, Eupatin displayed the highest binding affinity (−7.35 kcal/mol), exceeding that of the reference drug clavulanic acid (−4.99 kcal/mol), indicating a better structural complementarity with the enzyme's active site. The other phytocompounds exhibited moderate binding energies, ranging from −5.34 to −6.93 kcal/mol. This pattern in binding energies was consistent with the calculated inhibition constants (Ki).

3.2. Inhibition Constants (Ki)

The Ki values offer important insight into the inhibitory potency of the studied compounds. Eupatin (Ki = 4.11 μM) demonstrated superior activity compared to the reference inhibitor clavulanic acid (Ki = 220.94 μM), indicating its effectiveness at lower concentrations. Other phytocompounds, including Artemetin (8.64 μM), Casticin (18.31 μM), Chrysosplenetin (13.88 μM), Corymbosin (30.63 μM), and Ternatin (121.50 μM), also exhibited favorable inhibition constants within a promising range. Notably, all these compounds showed stronger inhibitory potential than the reference inhibitor.

3.3. Hydrogen bonds interactions

Hydrogen bonding plays a crucial role in stabilizing ligand–receptor complexes, with all analyzed ligands forming 4–9 hydrogen bonds within the active site. Eupatin showed the strongest binding, establishing nine hydrogen bonds with key residues such as SER70, SER130, ARG243, GLU166, and ASN170. Other phytocompounds, including Artemetin, Casticin, Chrysosplenetin, Corymbosin, and Ternatin, also demonstrated stable interactions through similar hydrogen bonding patterns. The reference inhibitor clavulanic acid formed six hydrogen bonds with important active site residues, showing comparable interactions with the selected phytocompounds.

3.4. Other molecular Interactions

Hydrophobic interactions play a key role in stabilizing ligand binding alongside hydrogen bonds. All flavonoid derivatives showed interactions with important residues, particularly Ala237 and Pro167, highlighting them as key binding hotspots. Eupatin interacted with Ala237, Pro167, and Val216, while Artemetin, Chrysosplenetin, and Corymbosin formed additional contacts with residues such as Tyr105 and Met270. Casticin and Ternatin also maintained stable hydrophobic interactions within the binding pocket. Despite fewer hydrophobic contacts, Eupatin exhibited the highest affinity, indicating that interaction quality and orientation are more important than quantity, while clavulanic acid showed only a single interaction with Ser235.

The ADMET analysis revealed that most compounds exhibited favorable pharmacokinetic properties. Eupatin, casticin, chrysosplenetin, corymbosin, and ternatin showed good bioavailability, whereas artemetin was predicted to be non-bioavailable. All compounds demonstrated adequate human intestinal absorption, indicating efficient uptake. The volume of distribution ranged from 0.44 to 0.60, suggesting moderate tissue distribution. Notably, all compounds except corymbosin were predicted as non-inhibitors of CYP3A4, indicating a lower risk of drug–drug interactions. Clearance values varied between 6.87 and 9.03, with eupatin showing the highest clearance rate. Furthermore, all compounds were classified under toxicity class 5 with high LD50 values, indicating low acute toxicity. Overall, eupatin exhibited the most balanced ADMET profile, supporting its potential as a promising lead compound.

3.6. Comparative Analysis with Calvulanic acid

All six flavonoid-derived phytocompounds demonstrated stronger binding to TEM-1 β-lactamase than the reference inhibitor, Clavulanic acid, which exhibited a binding energy of −4.99 kcal/mol and a Ki of 220.94 μM. Eupatin showed the highest affinity, with a binding energy of −7.35 kcal/mol and a Ki of 4.11 μM, stabilized by nine hydrogen bonds with key active site residues. Artemetin and Casticin displayed moderate inhibition (ΔG = −6.92 and −6.73 kcal/mol; Ki = 8.64 and 18.31 μM) through multiple hydrogen bonds and hydrophobic contacts. Chrysosplenetin, Corymbosin, and Ternatin exhibited weaker binding (ΔG = −6.63, −6.16, and −5.34 kcal/mol; Ki = 13.88, 30.63, and 121.50 μM), but they still had stronger binding and lower Ki than Clavulanic acid. These findings highlight the superior interaction profiles of phytocompounds, primarily driven by stronger hydrogen bonding and enhanced stabilization within the enzyme active site.

The present study highlights the potential of flavonoid-based phytocompounds as effective inhibitors of TEM-1 β-lactamase, a key enzyme responsible for bacterial resistance to β-lactam antibiotics. Among the selected phytocompounds compounds, Eupatin demonstrated the highest binding affinity, supported by a low inhibition constant (Ki = 4.11 μM). This strong interaction is linked to the formation of multiple hydrogen bonds and stable hydrophobic interactions within the enzyme's active site. In particular, the presence of hydroxyl and methoxy substituents appears to enhance binding by facilitating interactions with critical catalytic residues such as SER70, ARG243, and GLU166.

Comparative analysis with clinically used β-lactamase inhibitors, including Clavulanic acid, Sulbactam, and Tazobactam, further underscores the significance of these findings. Clavulanic acid, used as a reference compound due to its established clinical role, exhibited a considerably higher Ki value (220.94 μM), indicating weaker binding in docking

Table 1. Molecular docking results of the selected ligands, including binding energy, number of hydrogen bonds, hydrogen bond-forming residues, other interactions, and inhibition constants. These parameters describe the interaction pattern and binding strength of the ligands with the target protein. Overall, the table indicates that all selected compounds exhibit stable binding interactions and form multiple contacts with key amino acid residues, supporting their potential for further analysis.

Sl.No	Ligand	Binding energy (kcal/ Mol)	No. of Hydroge n Bonds	Hydrogen bond form residues	Other interactions
1	Eupatin	-7.35	9	SER70, ASN132, SER130,ARG243, ASN170, SER235, LYS73, GLU166, GLU239	ALA237, PRO167, VAL216,
2	Artemetin	-6.92	7	SER70, SER130, ARG243, ASN170, VAL216, GLU104, GLU166	PRO167, TYR105, ALA237, MET270,
3	Casticin	-6.73	6	SER70, SER130, ARG243, ASN132, SER235, VAL216	PRO219, TYR105, PRO167,
4	Chrysospleneti n	-6.63	6	SER70, SER130, ARG243, GLU166, GLU104, VAL216,	MET270, ALA237, TYR105, GLU104,
5	Corymbosin	-6.16	4	ARG243, SER70, ASN170, GLU166,	ALA237, ASN132, PRO167, VAL216
6	Ternatin	-5.34	7	SER70, ASN170, ALA237, SER130, TYR105, MET69, GLU104	TYR105, VAL216, ALA237
7	Clavulanic acid	-4.99	6	SER70, SER130, ARG243, ALA237, ASN170, SER235,	SER235

Table 2. Predicted ADMET parameters of the selected compounds, including logP, bioavailability, human intestinal absorption (HIA), volume of distribution (VDs), CYP3A4 inhibition, clearance, LD50, and toxicity class

Compound	logPapp	Bio availability	HIA	VDs	CYP3A4 inhibition	Clearance	LD50	Toxicity class
Eupatin	-4.71	Bioavailability	Absorbed	0.44	Non-Inhibitor	9.03	5000	5
Artemetin	-5.34	Non - bioavailability	Absorbed	0.48	Non-Inhibitor	6.87	5000	5
Casticin	-4.61	Bioavailability	Absorbed	0.48	Non-Inhibitor	8.49	5000	5
Chrysosplenetin	-4.68	Bioavailability	Absorbed	0.48	Non-Inhibitor	8.51	5000	5
Corymbosin	-4.65	Bioavailability	Absorbed	0.60	Inhibitor	7.53	4000	5
Ternatin	-4.55	Bioavailability	Absorbed	0.45	Non-Inhibitor	8.32	5000	5

simulations. The superior performance of Eupatin and other flavonoids suggests that these compounds may act as effective inhibitors. This contrasts with conventional inhibitors like Clavulanic acid, which typically rely on mechanism-based inhibition, mechanisms that are not fully represented in molecular docking studies. Therefore, the docking performance of flavonoids may reflect their ability to form stable interactions within the active site.

In addition to binding efficiency, pharmacokinetic and toxicity predictions provide further support for the therapeutic potential of these compounds. All selected flavonoids demonstrated favorable ADMET properties, including good absorption and low toxicity, as indicated by high LD_{50} values (4000–5000 mg/kg) and classification under toxicity class V, and another thing their predicted non-inhibitory effect on CYP3A4 suggests a lower risk of drug-drug interactions, which is an important consideration in drug development. In contrast, existing β-lactamase inhibitors, while effective, are often associated with ch allenges such as emerging resistance and variable pharmacokinetic profiles.

However, despite these promisi ng attributes, the ADMET analysis revealed relatively high clearance rates for the flavonoid compounds. This may affect their systemic bioavailability and duration of action, potentially reducing therapeutic effectiveness *in vivo*. Such limitations indicate the need for further optimization, possibly through structural modification or formulation strategies, to enhance their pharmacokinetic stability (Janakiramulu et al., 2025). Overall, the findings of this study suggest that flavonoid-based compounds, particularly Eupatin, represent promising lead candidates for the development of novel β-lactamase inhibitors. While conventional inhibitors such as Clavulanic acid remain crucial in clinical practice, the superior binding affinity, favorable safety profile, and reduced interaction liabilities of

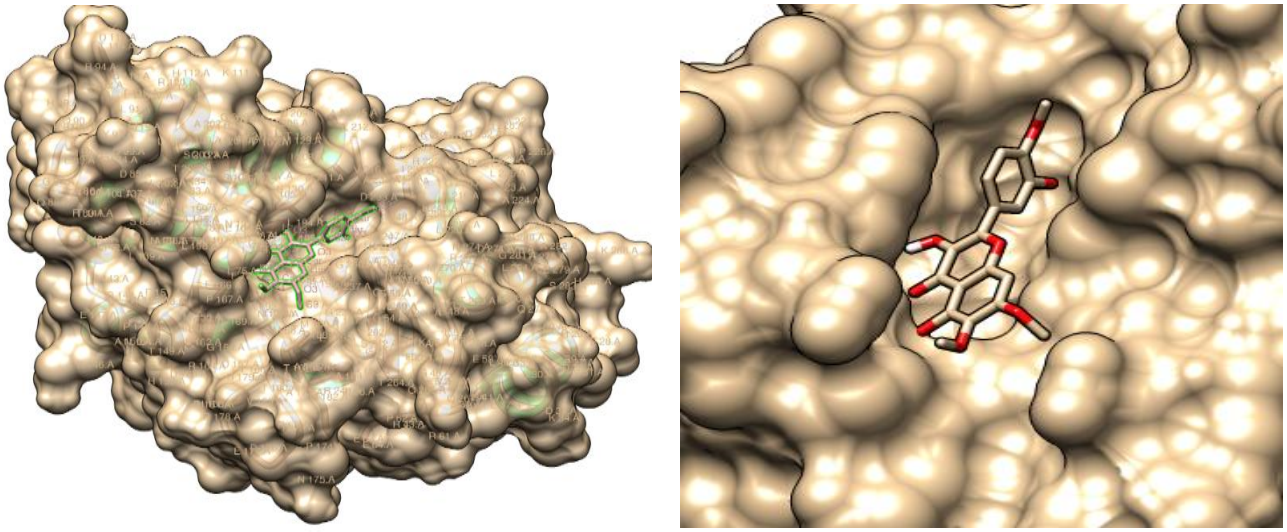

Fig 2. 3d representation of Eupatin binds with Tem 1 beta lactamases within the active site

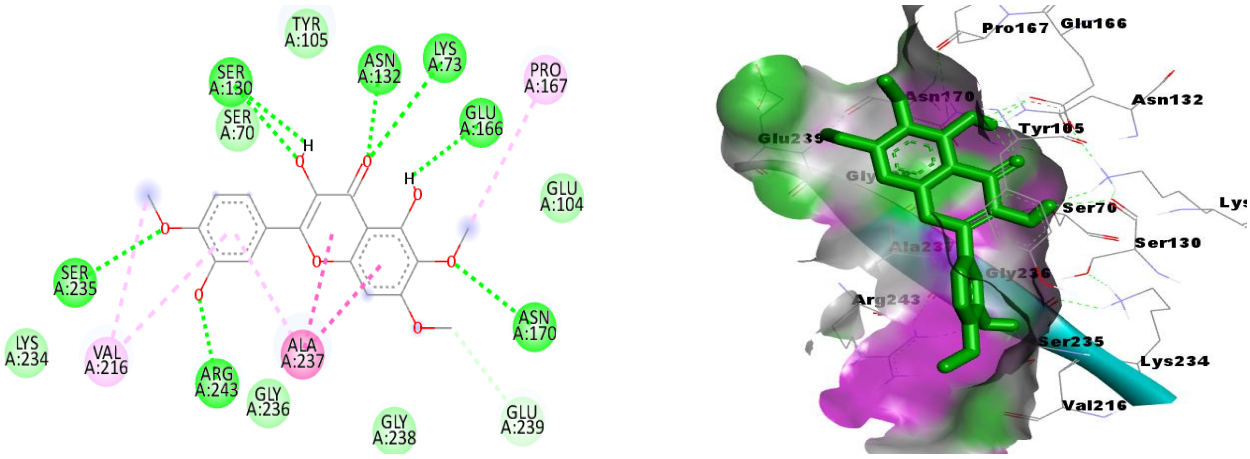

Fig 3. 2d representation of Eupatin binds with Tem 1 beta lactamases

these natural compounds provide a strong basis for further investigation. Future studies involving in vitro and in vivo validation will be essential to confirm their efficacy and advance their potential application in combating antibiotic resistance.

6. CONCLUSION

The present study highlights several flavonoid-derived phytocompounds as potential inhibitors of TEM-1 β-lactamase, with Eupatin identified as the most promising candidate due to its highest binding affinity and lowest inhibition constant. Its strong inhibitory potential, along with compounds such as Artemetin and Casticin, is attributed to multiple hydrogen bonds and stabilizing hydrophobic interactions with key active site residues, including SER70, ARG243, GLU166, and ASN170. These results suggest that flavonoid-derived scaffolds could serve as valuable leads for developing new TEM-1 β-lactamase inhibitors. Further experimental validation and structural optimization are warranted to explore their potential as therapeutic agents against β-lactam antibiotic resistance.

Competing interests:

The authors declare that they have no competing interests

References

[1] Banerjee, P., Kemmler, E., Dunkel, M., & Preissner, R. (2024). ProTox 3.0: a webserver for the prediction of toxicity of chemicals. Nucleic Acids Research, 52(W1), W513–W520.

[2] Bhatia, R. (2024). Environmental aspects of antimicrobial resistance in India: Current progress & way forward. Indian Journal of Medical Research, 159(1), 10–15.

[3] Binek, M., Kizerwetter-Świda, M., Rzewuska, M., Chrobak-Chmiel, D., & Sałamaszyńska-Guz, A. (2019). Antibiotic Resistance Of Bacteria A Growing Threat For Animals And Public Health. Postępy Mikrobiologii - Advancements of Microbiology, 58(3), 259–270.

[4] Cantón, R. (2014). Epidemiology and Evolution of Beta-Lactamases. In Evolutionary Biology of Bacterial and Fungal Pathogens (pp. 249–270). ASM Press.

[5] Daina, A., Michielin, O., & Zoete, V. (2017). SwissADME: a free web tool to evaluate pharmacokinetics, drug-likeness and medicinal chemistry friendliness of small molecules.

Scientific Reports, 7(1), 42717. https://doi.org/10.1038/srep42717
[6] Janakiramulu, P., & Mamidala, E. (2025). Molecular Docking and dynamic simulation analysis of flavonoid derivatives as COX-2 inhibitors. *In Silico Pharmacology*, *13*(2), 59.
[7] 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.
[8] Lipinski, C. A., Lombardo, F., Dominy, B. W., & Feeney, P. J. (2001). Experimental and computational approaches to estimate solubility and permeability in drug discovery and development settings 1PII of original article: S0169-409X(96)00423-1. The article was originally published in Advanced Drug Delivery Reviews 23 (1997) 3–25. 1. Advanced Drug Delivery Reviews, 46(1–3), 3–26.
[9] 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.
[10] Meng, X. Y., Zhang, H. X., Mezei, M., & Cui, M. (2011). Molecular docking: A powerful approach for structure-based drug discovery. Current Computer-Aided Drug Design, 7(2), 146-157
[11] 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.
[12] Namthabad, S., & Mamidala, E. (2014). Molecular docking of HIV-1 protease using alkaloids from tinospora cordifolia. *Int J Res Appl*, *1*(1), 12-6.
[13] Nandhini, P., Kumar, P., Mickymaray, S., Alothaim, A. S., Somasundaram, J., & Rajan, M. (2022). Recent Developments in Methicillin-Resistant Staphylococcus aureus (MRSA) Treatment: A Review. Antibiotics, 11(5), 606.
[14] Ness, S., Martin, R., Kindler, A. M., Paetzel, M., Gold, M., Jensen, S. E., Jones, J. B., & Strynadka, N. C. J. (2000). Structure-Based Design Guides the Improved Efficacy of Deacylation Transition State Analogue Inhibitors of TEM-1 β-Lactamase ,. Biochemistry, 39(18), 5312–5321.
[15] Pettersen, E. F., Goddard, T. D., Huang, C. C., Couch, G. S., Greenblatt, D. M., Meng, E. C., & Ferrin, T. E. (2004). UCSF Chimera—A visualization system for exploratory research and analysis. Journal of Computational Chemistry, 25(13), 1605–1612. https://doi.org/10.1002/jcc.20084
[16] Pires, D. E. V, Blundell, T. L., & Ascher, D. B. (2015). pkCSM: Predicting Small-Molecule Pharmacokinetic and Toxicity Properties Using Graph-Based Signatures. Journal of Medicinal Chemistry, 58(9), 4066–4072.
[17] 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.
[18] Ramana, B., & Chaudhury, A. (2012). Antibiotic resistance pattern of Pseudomonas aureuginosa isolated from healthcare associated infections at a tertiary care hospital. Journal of the Scientific Society, 39(2), 78. https://doi.org/10.4103/0974-5009.101850
[19] Rani, M. (2024). Mechanisms And Implications Of Antibiotic Resistance: A Global Public Health Challenge. Journal of Population Therapeutics & Clinical Pharmacology, 45–59. https://doi.org/10.53555/ztnsyy41
[20] Sachdev, C., Anjankar, A., & Agrawal, J. (2022). Self-Medication With Antibiotics: An Element Increasing Resistance. Cureus. https://doi.org/10.7759/cureus.30844
[21] Sharma, S., Jhalora, V., Mathur, S., & Bist, R. (2025). A Comparison of Antibiotics' Resistance Patterns of E. coli and B. subtilis in their Biofilms and Planktonic Forms. Infectious Disorders - Drug Targets, 25(2).
[22] Singh, N. (2023). Antibiotic resistance in healthcare associated infections: An Empirical Study causes and consequences. European Journal of Molecular & Clinical Medicine, 05(01).
[23] 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.
[24] Tacconelli, E., & Cataldo, M. A. (2008). Vancomycin-resistant enterococci (VRE): transmission and control. International Journal of Antimicrobial Agents, 31(2), 99–106.
[25] Tooke, C. L., Hinchliffe, P., Bragginton, E. C., Colenso, C. K., Hirvonen, V. H. A., Takebayashi, Y., & Spencer, J. (2019). β-Lactamases and β-Lactamase Inhibitors in the 21st Century. Journal of Molecular Biology, 431(18), 3472–3500.
[26] 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.
[27] Vyas, P. (2012). Antibiotic resistance pattern of mdr pathogens. IOSR Journal of Pharmacy (IOSRPHR), 2(5), 44–46.

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