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Research Article

Naringenin as a Promising Phytotherapeutic Scaffold Against MRSA: Molecular Docking and ADMET Profiling Study

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Abstract

Antimicrobial resistance has emerged globally, leading to the increased need to seek alternative therapeutic scaffolds to treat the Methicillin-resistant Staphylococcus aureus (MRSA). Naringenin, a flavanone compound of citrus fruit, has antimicrobial and anti-inflammatory effects, and thus is a potential phytotherapeutic agent. The aim of the study was to determine the potential of naringenin to interact with MRSA at the molecular level as well as have a pharmacokinetic profile through molecular docking and in silico ADMET analysis. The molecular docking of the MRSA target protein (PDB ID: 6O9S) to assess binding affinity and patterns of interaction was performed using UCSF Chimera. Clindamycin was taken as a standard. The profiles of ADMET were determined on ADMETlab 3.0 and ADMET-AI to evaluate the drug-likeness, absorption, distribution, metabolism, excretion and toxicity parameters. Naringenin had a higher binding affinity (-7.5 kcal/mol) than clindamycin (-6.5 kcal/mol). Analyzing of interaction indicated optimization of hydrophobic interaction with key residues (PHE423, TRP426, TYR438, ILE533, GLY530) and stable hydrogen bonding especially with TRP426. The compound had a lower molecular flexibility which implied a lower entropic penalty when binding. The analysis of ADMET made it possible to verify adherence to the Rule of Five developed by Lipinski, high absorption in the intestine (0.95), good Caco-2 permeability, and minimal chances of hepatotoxicity and cardiotoxicity. Nonetheless, the predictions borderline mutagenicity and genotoxicity suggest that more experiments are required. Naringenin has good binding stability and good pharmacokinetic properties in comparison to the normal antibiotic, which indicates a promising candidate of phytotherapeutic scaffold to develop anti-MRSA drugs.

Keywords: Naringenin; Methicillin-resistant *Staphylococcus aureus* [MRSA]; Molecular Docking; ADMET; Phytotherapeutic; Antimicrobial Resistance.

1. INTRODUCTION

Antimicrobial resistance (AMR) is turning out to be one of the most urgent world health issues in the 21st century. The Methicillin-resistant Staphylococcus aureus (MRSA) is one of the primary clinical problems in regard to the presence of resistance to b-lactam antibiotics and resistance to many other antimicrobials[1]. The scope of MRSA infections is broad where mild skin and soft tissue infection occurs in one extreme and on the other extreme severe infections like pneumonia, endocarditis, osteomyelitis, and septicaemia are life-threatening. Adaptability of the pathogen, its capacity to form biofilm, and resistance determinants acquisition like the mecA gene in penicillin-binding protein 2a (PBP2a) are major limitations to therapy[2,3]. Although the use of last-resort antibiotics like vancomycin and linezolid is available, the rising number of diminished susceptibility and resistance to treatment is a simulation of the necessity of new antibacterial agents with alternative mechanisms of action[4].

Natural products have been traditionally an abundant source of therapeutic agents, especially in the area of anti-infectives. Phytochemicals provide structural diversity, excellent safety, and ability to regulate numerous biological targets. Flavonoids have received so much attention among them in terms of antimicrobial, antioxidant, anti-inflammatory, and anti-cancer effects[4,5]. Naringenin is a naturally occurring flavanone that is mostly found in citrus fruits like grapefruit and oranges; this is coming as a promising bioactive scaffold. Naringenin, structurally a 4', 5, 7-trihydroxyflavanone, is capable of a variety of pharmacological actions such as antibacterial against Gram positive as well as Gram negative pathogens[6].

A number of studies have shown that naringenin is able to destroy the integrity of bacterial cell membrane,

quorum sensing, and virulence factor expression. Flavonoids have been found to prevent cell wall biosynthesis and DNA replication by interfering with key enzymes and proteins in Gram-positive bacteria and especially Staphylococcus aureus. Such complex systems render naringenin as an attractive agent to counter MRSA and other resistant strains. Additionally, it has a natural origin and is comparatively less cytotoxic, and this is an advantage it possesses in the context of further structural optimization[4,7,8]. Computational drug discovery methods have in recent years increased pace in the identification and optimization of novel antimicrobial agents. Molecular docking has been found to offer useful information about ligand-target interactions, binding affinities and the possible inhibitory pathways at the atomic levels. The in silico determination of the drug-likeness and safety by ADMET (Absorption, Distribution, Metabolism, Excretion, and Toxicity) profiling of the drug also helps in predicting the pharmacokinetic and toxicity parameters. A hybrid approach of molecular docking and ADMET analysis provides an economical and time-saving method of screening phytotherapeutic candidates on validated bacterial targets like PBP2a, DNA gyrase and dihydrofolate reductase[5,9–13].

The increasing challenge of MRSA and the constraints of current antibiotics make plant-derived scaffolds an appealing target in the field of antimicrobial drug discovery. Computational methods of scaffold discovery are a logical approach to this challenge, and exploration of the plant-derived scaffolds is rational. Thus, the current paper will explore molecular interactions of naringenin with the selected MRSA protein targets by docking simulations and assess pharmacokinetic and toxicity properties of naringenin by using a set of ADMET predictions. This study aims to use naringenin as a promising phytotherapeutic scaffold by elucidating its binding potential and drug-like properties in the creation of new anti-MRSA agents[9,11].

2. MATERIALS AND METHODS

2.1. Selection of Phytochemicals for Wound Healing

The selection of Naringenin (4′,5,7-trihydroxy-flavanone), a prominent dietary trihydroxyflavanone found primarily in citrus species, was based on its established multi-therapeutic profile encompassing potent antioxidant, anti-inflammatory, and antimicrobial properties.

2.2. Selection of the target for antibacterial

The selection of the molecular target was grounded in a comprehensive literature review identifying critical virulence factors in methicillin-resistant *Staphylococcus aureus* (MRSA) that impede cutaneous wound healing. The selection of methicillin-resistant *Staphylococcus aureus* (MRSA) as the primary antibacterial target was predicated on its clinical prevalence as a leading cause of chronic, non-healing wound infections. MRSA actively impedes the physiological stages of wound repair by forming dense biofilms and secreting exotoxins that provoke a prolonged inflammatory response, ultimately leading to tissue necrosis and inhibited re-epithelialisation [14].

2.3. Molecular docking

Molecular docking was performed to identify potential lead compounds and to understand their binding interactions with the target protein. Docking of the selected compounds was performed to determine their binding modes within the target protein's active site (PDB ID: 6O9S). The crystal structure of the protein (PDB ID: 6O9S) was retrieved from the RCSB Protein Data Bank (PDB)[15]. The protein structure was prepared for docking using UCSF Chimera. All solvent molecules and unnecessary heteroatoms were removed, and hydrogen atoms were added to the cleaned protein structure. The prepared protein was then saved in PDB format for subsequent docking studies. The grid box was defined with its centre at X = 67.5855, Y = 78.9792, and Z = 152.208, with grid dimensions of 24.9666 x 21.9024 x 22.1633. After completion of the docking process, the binding poses and interaction profiles of the protein–ligand complexes were analysed using 2D and 3D visualisation tools available in UCSF Chimera.

2.4. ADMET of phytochemical

For a robust pharmacokinetic and safety profile for the lead compounds, a dual-server computational analysis was conducted using ADMET lab 3.0[16] and ADMET-AI. Physiochemical and Medicinal Chemistry Viability of Molecules. Medicinal chemistry viability was determined by employing PAINS and BMS filters to screen for structural alerts and utilising Lipinski's Rule of Five to assess drug-likeness of the molecules. In addition to medicinal chemistry viability, absorption characteristics of the molecules were evaluated by determining the compounds' Caco-2 permeability and HIA (Human Intestinal Absorption) values, along with distribution factors of PPB (Plasma Protein Binding) and BBB (Blood-Brain Barrier) penetration. Furthermore, metabolic profiles of the molecules were evaluated to determine their potential as substrates or inhibitors of major cytochrome P450 (CYP450) isoforms, as well as to assess their potential for excretion via clearance rates and half-life predictions. Finally, toxicity assessments of the molecules were performed to predict the potential for adverse effects (mutagenicity, hERG channel inhibition, DILI [Drug-Induced Liver Injury], and LD50 [acute oral toxicity]) and to ensure that the phytochemicals employed are safe and compatible with wound healing applications. Admet-Ai was also used to compare the molecules' percentile rankings against Drug Bank-approved therapeutics and to determine the potential of the phytochemicals to interfere with endocrine disruption and stress response pathways, thereby confirming that the molecules meet safety standards for use in wound healing applications. [17, 18]

3. RESULT AND DISCUSSION

3.1. Molecular docking visualization

A thermodynamic advantage in naringenin over the clinical standard clindamycin in the target binding

pocket was observed at its binding affinity of about 7.5 kcal/mol-1, as compared to 6.5 kcal/mol-1 in the molecular docking analysis, respectively. This difference in binding energy implies that naringenin has a stronger shape complementarity and electronic compatibility with the active site and therefore forms a more stable ligand-protein complex. A structural explanation of this discrepancy is achieved by a critical analysis of the molecular flexibility properties, with clindamycin having ten active torsional degrees of freedom as compared to four in naringenin. The increased rotational freedom of the clindamycin molecule, as compared to that of a solution, would carry a large entropic penalty on the binding process since the molecule must be in a flexible form in solution and then adopt a constrained, bioactive form on binding. The lesser flexibility of naringenin, on the contrary, reduces this entropic loss, and the binding energy can be better converted into a desirable affinity score. The interaction profile indicates that the hydrophobic core of the two ligands is conserved and involves PHE423, TRP426, TYR438, ILE533, and GLY530, which is an indication that the hydrophobic pocket is a primary determinant in anchoring the two ligands. Nonetheless, subtle variations of the polar and H-bond structures display different binding modes. Both ligands use a high density of polar residues, such as SER439, SER391, ASN390, THR531, and ASN441, but clindamycin uses another one, ASN478, and interacts with a positively charged LYS394, which is not present in the naringenin complex. Other contacts notwithstanding, the overall binding energy of clindamycin is still lower. This observation suggests that individual interactions, including the mutual side-chain H-bond with TRP426, are better optimised in naringenin, or that the steric fit of the flavanone skeleton of naringenin is better at pushing high-energy water molecules out of the hydrophobic cavity. Moreover, the two molecules form interactions with the negatively charged residues GLU425 and GLU538, thus forming a balanced electrostatic environment. The absence of interaction between naringenin and LYS394 indicates that it does not bind as much to basic amino acids, which could allow it to have a pharmacological benefit in the environment with variable pH conditions, where lysine protonation forms can change. More importantly, data suggest that the high potency of naringenin in silico is the result of a synergistic interaction of reduced internal strain, accurate H-bond positioning of relevant residues (TRP426) and effective exploitation of the hydrophobic sub-pockets. Although the range of polar contacts of clindamycin is broader, the increased flexibility of its molecular structure probably leads to an increased valence of the binding mode, which is not as energetically favourable as the locked orientation of naringenin. The results indicate that naringenin would be a stronger structure to use in the further optimisation of leads, as it can bind with high affinity by having a more efficient interaction-to-atom-count ratio compared to the larger and more flexible standard antibiotic. Therefore, the comparative docking research highlights the promise of naringenin as an effective inhibitor, which is better than the standard due to better thermodynamic stability and ideal spatial orientation in the catalytic or allosteric site of the target protein. [19,20]

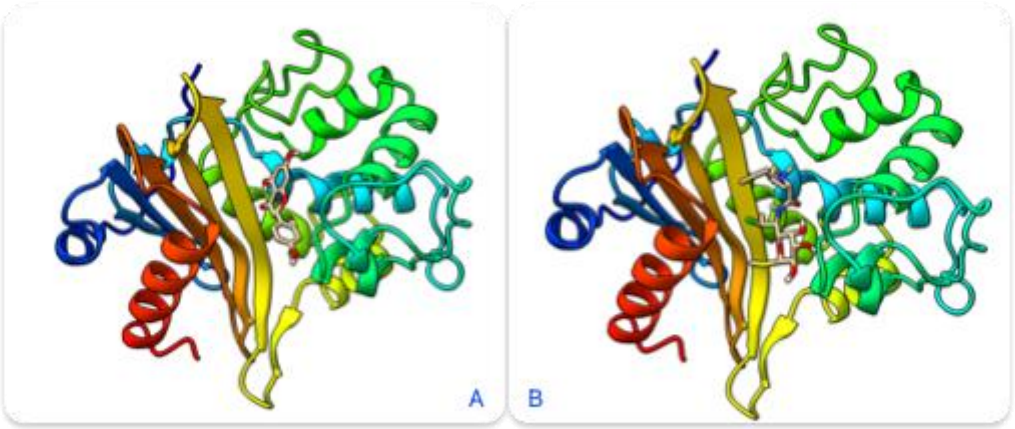

Figure 1. 3D interaction of Naringenin vs Clindamycin; (A) Naringenin, (B) Clindamycin

Figure 2. Residue index of 3D diagram: (A) Residue interaction of naringenin, (B) Residue interaction of clindamycin.

Table 1: Molecular docking analysis of Naringenin and Clindamycin against the MRSA target (PDB ID: 6O9S)

Parameter	Naringenin	Clindamycin (Standard)
Best Binding Affinity	-7.5 kcal/mol	-6.5 kcal/mol
Molecular Flexibility	4 Active Torsions	10 Active Torsions
Hydrophobic Interactions	PHE423, TRP426, TYR438, ILE533, GLY530	TYR438, PHE423, TRP426, ILE533, GLY530
Polar/H-Bond Interactions	SER439, SER391, ASN390, THR531, ASN441	SER439, ASN441, ASN390, SER391, THR531, ASN478
Negatively Charged Residues	GLU425, GLU538	GLU425, GLU538
Positively Charged Residues	None identified	LYS394
Specific H-Bonds	Sidechain H-bond with TRP426	Sidechain H-bond with TRP426

3.2. ADMET

The ADMET (Absorption, Distribution, Metabolism, Excretion, and Toxicity) screening of naringenin and the reference drug clindamycin provides a comprehensive comparative evaluation of their pharmacokinetic characteristics, resulting in the fact that both compounds possess good drug-like properties, and naringenin has significantly high potential to be an oral lead. Naringenin fulfils the Lipinski Rule of Five, with a molecular weight of 272.07, predicted to be the most abundant in the body as well as in drug absorption within the body, through a logP of 2.59. Comparatively, clindamycin also fits the requirements, although it has a molecular weight more equal to 424.18 Da and a logP of 2.66, which is closer to the limit of the molecular weight provided by the Lipinski rule. The Caco-2 permeability value of 4.98 log cm s^{-1} was compared with that of clindamycin of 5.35 log cm s^{-1}, which indicates the more effective passive ingestion across the intestinal epithelium due to its lighter molecular mass and smaller size. The naringenin has a topological polar surface area (TPSA) of 86.99 $Å^2$, which was lower than 140 $Å^2$, further supporting its positive oral bioavailability, but the TPSA of clindamycin is 102.26 $Å^2$, which was higher but still within the acceptable range of drug development candidates. Naringenin and clindamycin have high potentials of human intestinal absorption (HIA), such as 0.95 and 0.86, respectively. Concerning distribution, both compounds fail to pass through the blood-brain barrier (BBB), with naringenin having a permeability of 0.00015 and clindamycin 0.000035, which implies an insignificant central nervous system (CNS) toxicity risk or unwanted toxicity. However, there was a very significant difference in the plasma protein binding (PPB); naringenin has a binding affinity of 94.98, and clindamycin has an affinity of 73.15. This increase in PPB can affect the amount of distribution and increase the systemic circulation of naringenin, which can lead to the extension of the period of action, although it may also lead to a decrease in the percentage of free, active drug at the target site. The computed value of the volume of distribution at steady state (VDss) of naringenin is known as a log of 0.011, which was relatively large in terms of tissue distribution, and it was slightly higher than the value of 0.11 that was predicted to be that of clindamycin. Metabolically, the two compounds have a low likelihood of CYP3A4 inhibition (probability <0.01), thus indicating that there was very low potential for drug-drug interactions because of other agents by the same primary metabolism. Conversely, naringenin exhibits a significantly greater likelihood of inhibiting CYP2C9 (0.94) and CYP1A2 (0.58), and when used with the enzyme, it should be treated with excellent caution since it may cause inhibition of clindamycin but with little effect on all the enzymes of the cytochrome P450 family. Regarding excretion, the half-life of naringenin was estimated to be 1.31 h, and its plasma clearance is 6.89 L h^{-1} kg^{-1}, with a relatively high drug clearance compared to clindamycin, which has a half-life of 1.67 h and a clearance rate of 3.33 L h^{-1} kg^{-1}. The toxicological profile reveals that naringenin has a sufficiently safe profile based on low predicted hERG inhibition and drug-induced liver injury and risk, as well as cardiotoxicity predicted by hERG, as well as a clinical reference, clindamycin, showing liver safety (DILI=0.11) and a low risk of cardiotoxicity (hERG=0.04). One of the issues to note is that naringenin has a borderline Ames mutagenicity score (0.52), which suggests a possibility of being mutagenic, which could only be confirmed through in vitro studies, but clindamycin is strictly negative (probability = 0.014). In addition, the predicted genotoxicity (0.97) and carcinogenicity (0.59) of naringenin indicate that further safety studies are required, especially in comparison with the lower risk scores of clindamycin in the two areas of toxicity. The probability of skin sensitisation was also higher in naringenin (0.74) as compared to clindamycin, although both are usually considered safe. Interestingly, the naringenin was expected to verify the P-glycoprotein (P-gp=0.95), which probably increases the absorption of co-administered medications and puts the risk of toxicity build-up; however, clindamycin is rather a P-gp substrate. Altogether, the ADMET profile of naringenin remains highly competitive with the clinical standard clindamycin, especially in terms of intestinal permeability, and meets all the drug-likeness criteria, but its long-term safety, especially in terms of genotoxicity and mutagenicity, should be investigated. Finally, comparative data support naringenin as a

promising pharmacophore to exploit in therapeutic development, as it has a pharmacokinetic profile that equals and, in some absorption-related parameters, it surpasses the well-known antibiotic clindamycin. [21,22].

Table 2. Comparative ADMET Profiling of naringenin and clindamycin.

Property	Naringenin (Result)	Clindamycin (Standard)	Range
Molecular Weight	272.07 Da	424.18 Da	Both within range (< 500)
logP (Lipophilicity)	2.59	2.66	Optimal hydrophobicity
logS (Solubility	-4.02	-4.10	Moderately soluble
TPSA	86.99	102.26	Good oral bioavailability (< 140)
Lipinski Rule Violations	0	0	Both are drug-like
HIA (Absorption)	0.95 (High)	0.86 (High)	Excellent oral absorption
Caco-2 Permeability	-4.98 (High)	-5.35 (Moderate)	Naringenin is more permeable
BBB Permeability	0.00015 (Low)	0.00000 (Low)	No CNS side effects
Plasma Protein Binding	94.98%	73.15%	Naringenin highly bound
CYP3A4 Inhibition	0.01 (Negative)	0.005 (Negative)	Low drug-interaction risk
Mutagenicity	0.52 (Borderline)	0.014 (Negative)	Clindamycin is safer
DILI (Liver Toxicity)	0.32 (Low)	0.11 (Low)	Both are liver-safe
hERG Inhibition	0.10 (Low)	0.04 (Low)	Low cardiotoxicity risk

4. CONCLUSION

The present study has methodically assessed naringenin as a prospective phytotherapeutic scaffold on MRSA through molecular docking and overall ADMET profiling methodologies. The docking study showed that naringenin had better binding affinity than the clinical benchmark clindamycin, which is confirmed by the optimum hydrophobic interactions and stable hydrogen bonding in the active site of the MRSA target protein (PDB ID: 6O9S). The drawback of its molecular flexibility meant that entropic penalties were less, which led to increased thermodynamic stability of the ligand protein complex. The ADMET analysis also supported the drug-like capabilities of naringenin, demonstrating that it is highly compliant with the Rule of Five, has no problems with intestinal absorption, has excellent membrane permeability, low hepatotoxicity, and no significant cardiotoxic liability. Though the results had predicted borderline mutagenicity and genotoxicity, these results should merely be further validated in in vitro and in vivo experiments and not to nullify its therapeutic success. Altogether, molecular docking and pharmacokinetic prediction combined allow us to assume that naringenin is an efficient and structurally viable pharmacological scaffold that may be further used as an anti-MRSA drug. Further experimental validation and structural optimisation may enhance its safety and potency profile.

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Author Contributions

V.K.Y. conceptualized the study and performed molecular docking analysis. V.S. conducted ADMET profiling and data interpretation. V.K. assisted in data validation and manuscript drafting. A.V. supervised the research work, reviewed the manuscript critically for intellectual content, and approved the final version for submission.

Conflict of Interest: The authors declare that there is no conflict of interest regarding the publication of this research work.

Competing Interests: The authors declare that they have no competing financial or non-financial interests related to this work.

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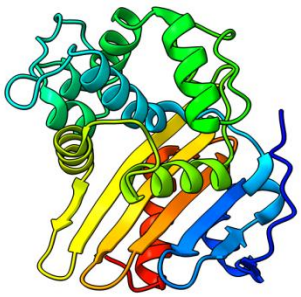
Protein: id 6SO9



Ligand: Naringenin