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Targeting Fibronectin-Binding Proteins in *Staphylococcus aureus*: *In Silico* Discovery and Pharmacokinetic Profiling of Novel Anti-Adhesion Inhibitors

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Abstract

Staphylococcus aureus is a highly virulent Gram-positive bacterium of global concern due to its potent virulence factors and antimicrobial resistance, causing severe morbidity and mortality. This study employed an anti-virulence approach that targets Fibronectin-binding proteins (FnBPs), key adhesion and immune-evasion determinants, offering an alternative to traditional antibiotics. Computational screening identified inhibitors, including Benzoic Acid (−8.61 kcal/mol), ML346 (−7.34 kcal/mol), and Gallic Acid (−7.29 kcal/mol), which showed strong binding potential. ADMET profiling confirmed good pharmacokinetic behavior and compliance with Lipinski's Rule of Five. These results highlight small-molecule inhibitors of FnBPs that can disrupt a major virulence pathway in *S. aureus*, providing a computational foundation for anti-virulence therapy development requiring experimental validation.

Article highlights

What is already known

- *S. aureus* is a leading human pathogen with increasing multidrug resistance.
- Fibronectin-binding proteins (FnBPs) are essential virulence determinants for adhesion, biofilm formation, and immune evasion.
- In silico methods like molecular docking and ADMET profiling are extensively employed in the identification and optimization of drug-like molecules.

What this paper adds

- Identifies strong binding pockets in FnBPs of *S. aureus* using structure-based docking.
- Reports novel inhibitors (e.g., Benzoic acid, ML346, Gallic acid, Isovitexin, Quercetin) with binding energies up to −8.61 kcal/mol.
- Establishes FnBPs as promising anti-virulence therapeutic targets to combat antibiotic-resistant *S. aureus*.

Introduction

S. aureus is a highly adaptable, Gram-positive bacterium that is a significant global health threat as a commensal organism and an opportunistic pathogen (Tong et al., 2015). It can colonize the cutaneous and mucosal surfaces of healthy people, but in conditions that are favorable, it becomes a pathogen that causes a broad range of infections. These vary from superficial lesions, e.g., impetigo, folliculitis, and abscesses, to invasive and life-threatening infections like pneumonia, endocarditis, osteomyelitis, septicemia, and toxic shock syndrome (Lowy, 1998; Turner et al., 2019). Its epidemiological significance is further amplified by the emergence of methicillin-resistant *S. aureus* (MRSA), which has made standard β-lactam treatments ineffective (Lee et al., 2018). This escalating resistance

reflects the need for novel approaches against *S. aureus* infections outside of the classical antibiotic strategy (Masumi et al., 2022; Ul-Haq et al., 2023).

The virulence of *S. aureus* is due to a vast array of virulence factors such as toxins, enzymes, and cell surface proteins involved in host invasion, colonization, and immune evasion (Foster et al., 2014). Of these, microbial surface components recognizing adhesive matrix molecules (MSCRAMMs) are of special significance. These proteins mediate adhesion of bacteria to extracellular matrix molecules like fibronectin, fibrinogen, and collagen, thus supporting colonization and persistence of infection (Schroeder et al., 2009). One of the prominent members of this group is the fibronectin-binding protein (FnBP), which plays a pivotal role in supporting the attachment of *S. aureus* to host tissues (Keane et al., 2007).

FnBPs are big, multi-functional proteins that facilitate not only fibronectin adhesion but also internalization of bacteria into non-phagocytic host cells via fibronectin–integrin bridges (Sinha et al., 2000). After being internalized, *S. aureus* can survive intracellularly, protected from immune elimination and many antibiotics (Kubica et al., 2008). This capacity is one of the causes of chronic and recurrent infections, especially in relation to indwelling medical devices where biofilm development is common (Arciola et al., 2012). Biofilms, complex communities of bacteria embedded in extracellular polymeric substances, impart a further level of resistance to

antibiotics and immune responses, rendering device-related infections very hard to eliminate. Overall, FnBPs are not only essential for colonization and persistence but also for enabling the transition of *S. aureus* from a commensal to an invasive pathogen (Liu et al., 2024).

Traditional treatment approaches mainly depend on antibiotics like vancomycin, linezolid, and daptomycin; however, multidrug-resistant strains undermine their long-term effectiveness (Chambers & DeLeo, 2009). Further, due to FnBP-mediated adhesion and biofilm structure, *S. aureus* can escape from host immunity and antimicrobial penetration, and thus antibiotic monotherapy proves insufficient for complete elimination. This scenario puts into view a significant therapeutic deficit and the urgency for alternative strategies that specifically hit bacterial virulence over viability. Anti-virulence measures, which incapacitate pathogens without exerting strong selective pressure for resistance, offer a compelling alternative in contemporary drug research (Cegelski et al., 2008).

Targeting FnBPs is particularly attractive, as inhibition of these proteins would impair bacterial adhesion, colonization, and invasion, thereby attenuating infection severity while leaving commensal flora largely unaffected. Recent advances in structural biology and computational chemistry provide an unprecedented opportunity to exploit FnBPs as drug targets. With the possibility of crystallographic data, structure-based drug design could be applied to find small molecule binders that cross-react with FnBP binding sites (Keane

et al., 2007). By inhibiting the fibronectin-binding domain, these inhibitors would be able to preclude *S. aureus* from attaching to host tissues, preventing initial infection and biofilm formation (JAYS & Saravanan, 2024, Verma & Chouhan, 2018a).

S. aureus remains a significant challenge owing to its ability to adapt, develop resistance, and employ virulent strategies. FnBPs as multi-functional mediators of adhesion and invasion are an attractive anti-virulence target. By means of bioinformatics-guided methods such as molecular docking and ADMET analysis, the present study will lay the groundwork for the rational design of inhibitors of FnBP (Kumar Suryawanshi et al., 2022). Eventually, such approaches could lead to new therapeutics that disempower *S. aureus* pathogenicity, improve host defenses, and minimize dependence on traditional antibiotics.

Materials and Methods

This research employed an *in silico* approach to investigate small-molecule inhibitors of the fibronectin-binding proteins (FnBPs) of *S. aureus*. The *in silico* work flowas shown in Figure 1. involved sequential stages of protein and ligand preparation, molecular docking, virtual screening, and pharmacokinetic screening to detect and screen candidate molecules (Pandey et al., 2017, Verma & Chouhan, 2018b).

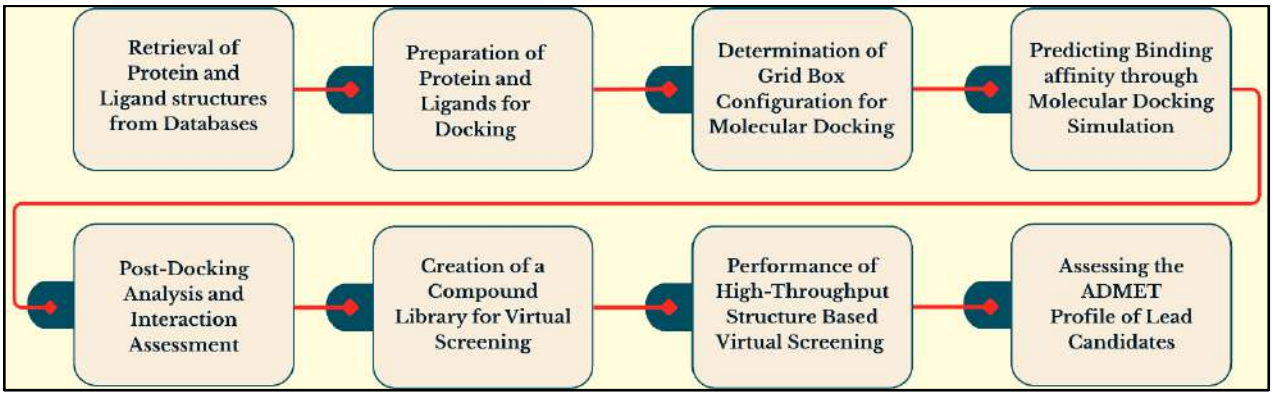

Figure 1: Flowchart of the Structure-Based Drug Discovery

Computational Environment and Software

The computational simulations were run on an ASUS VivoBook laptop with a 12th Gen Intel(R) Core (TM) i5-12500H 2.50 GHz processor and 16.0 GB RAM. Ubuntu 24.04.2 LTS Linux distribution was used under Windows Subsystem for Linux (WSL) to execute high-throughput virtual screening and molecular docking simulations.

Target Protein and Ligand Preparation

This paragraph explains the preparatory procedures necessary for both the FnBPs target protein and the chosen small-molecule ligands.

Target Protein Recovery and Preparation

The three-dimensional crystal structure of *S. aureus* Fibronectin-binding proteins (FnBPs) with PDB ID: 2RKY, as presented in Figure 2, was downloaded from the RCSB Protein Data Bank. The PDB file from the unrefined state was prepared for docking with AutoDockTools 1.5.7. Preparation involved the elimination

of crystallographic water molecules and non-essential heteroatoms from the structure. Polar hydrogen atoms were subsequently added, and Kollman united-atom charges were imposed to consider electrostatic interactions in physiological conditions. The receptor structure that had been completely prepared was stored in PDBQT format for further docking studies.

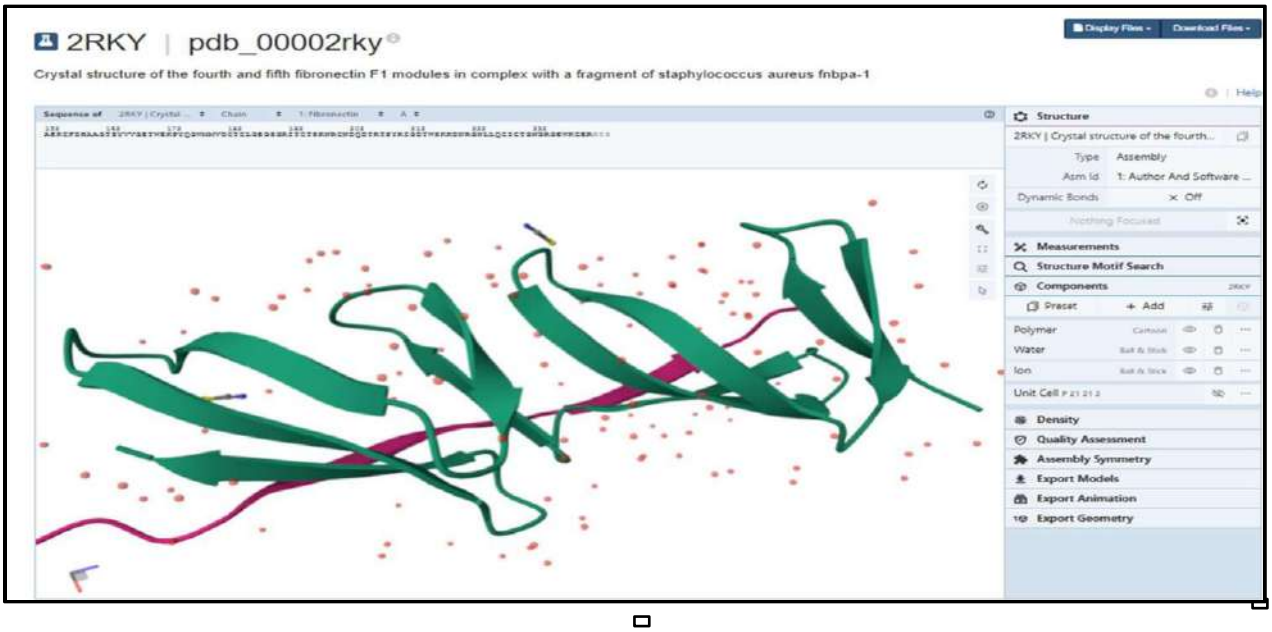

Figure 2: 2RKY Structure in Protein Database (PDB)

Ligand Selection and Preparation

Seven small-molecule ligands that have been reported or potentially have antimicrobial and anti-virulence activity were chosen for docking analysis. These were ML346 (CID: 767276), Isovitexin (CID: 162350), Gallic Acid (CID: 370), Quercetin (CID: 5280343), Triazolothiadiazole (CID: 136376653), Morin (CID: 5281670), and Benzoic Acid (CID: 82546561), as indicated in Figure 3. The three-dimensional structures of the ligands were downloaded from the PubChem

database in SDF format. Each ligand was converted from SDF to PDB format using Open Babel, protonated, optimized for geometry, and energy minimized with the MMFF94 force field (O'Boyle et al., 2011). To provide docking flexibility, Gasteiger charges were assigned, and rotatable bonds were set using AutoDockTools 1.5.7. The ligands fully prepared were saved as PDBQT format to be compatible with docking simulations.

Molecular Docking Protocol

A step-by-step two-stage molecular docking protocol was utilized to make predictions on the binding affinity and pattern of interaction of the chosen ligands against the FnBPs protein. Blind docking in AutoDock version 4.2 was conducted in the initial step to search the entire surface of the protein and identify putative binding sites. A big grid box covering the entire receptor structure was set to guarantee unbiased identification of the binding sites. At the second stage, the top-scored binding site of the blind docking was optimized using targeted docking, where a smaller grid box was placed on the previously located active pocket to get better ligand orientations and binding affinities. Docking simulations were performed using the Lamarckian Genetic Algorithm (LGA) with multiple independent runs for reproducibility. Binding affinities (given in kcal/mol) and conformational clusters were examined to find the most favorable protein–ligand complexes (Trott & Olson, 2010).

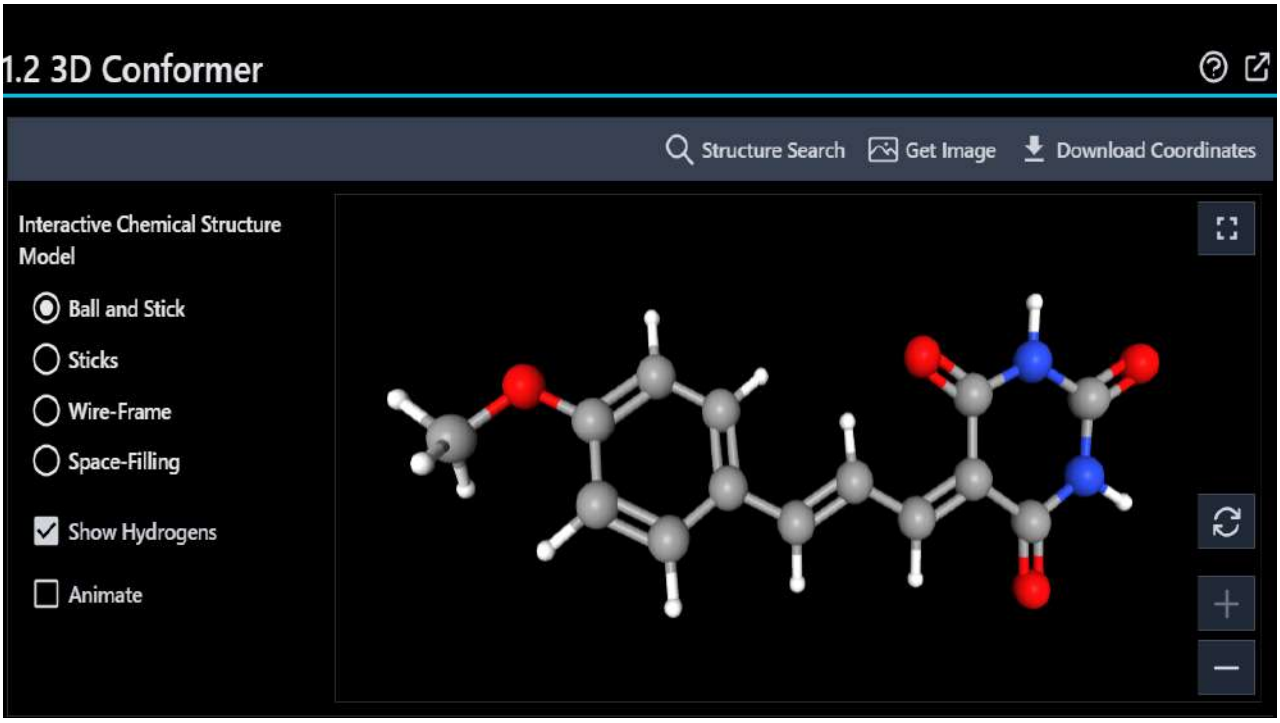

Figure 3. Chemical structure of ML346 (CID 767276) from PubChem

Blind Docking

At the initial stage, blind docking was used to scan the entire FnBPs protein surface for possible ligand-binding cavities. A 126 Å × 126 Å × 126 Å grid box was centered on the protein coordinates with a 0.850 Å spacing to cover the entire structure. Docking simulations were executed using the Lamarckian Genetic Algorithm (LGA) in AutoDock 4.2 to facilitate an unbiased search of potential binding sites throughout the FnBPs structure.

Focused Docking

With the results of blind docking, a probable binding pocket with high probability was selected and identified for detailed docking analysis. A 100 × 100 × 126 Å smaller grid box with a spacing of 0.900 Å was placed on the coordinates of the most optimal binding site for each ligand,

which are given in Table 2. The focused docking strategy permitted a more accurate estimation of binding affinities and offered insight into the orientation and interaction patterns of the chosen ligands inside the active site of FnBPs.

Visualization of Interactions

The docking outcomes for top-scoring compounds were examined with AutoDockTools 1.5.7 to determine their interaction patterns inside the FnBPs binding pocket. Hydrogen bonds, hydrophobic interactions, π-stacking, and other non-covalent ligand–amino acid residue interactions were attentively screened. Visualization allowed identification of crucial residues involved in ligand binding and stability inside the active site.

Virtual Screening

To expand the search for novel inhibitors, a high-throughput virtual screening was conducted using AutoDock Vina. For each of the seven initially selected compounds, a library of 1000 structurally similar analogs was retrieved from the PubChem database. These libraries were filtered according to Lipinski's Rule of Five, which evaluates molecular weight, logP (lipophilicity), hydrogen bond donors, and hydrogen bond acceptors, thereby ensuring drug-likeness (Bandyopadhyay et al., 2016). Only structures that met these conditions were shortlisted for docking into the FnBPs active site defined under focused docking (Morris et al., 2009).

ADMET and Physicochemical Evaluation

The highest-ranking compounds of virtual screening, determined according to their binding energies, were then assessed further for pharmacokinetic and physicochemical traits. The SMILES of the chosen molecules were entered into the SwissADME web server to predict ADMET (Absorption, Distribution, Metabolism, Excretion, and Toxicity) profiles (Daina et al., 2017). Important properties as shown in Figure 4. were examined which includes gastrointestinal absorption, blood–brain barrier permeability, cytochrome P450-mediated metabolism, solubility, synthetic accessibility, and drug-likeness filters. This profiling allowed the compounds with good bioavailability and safety to be picked for potential therapeutic applications.

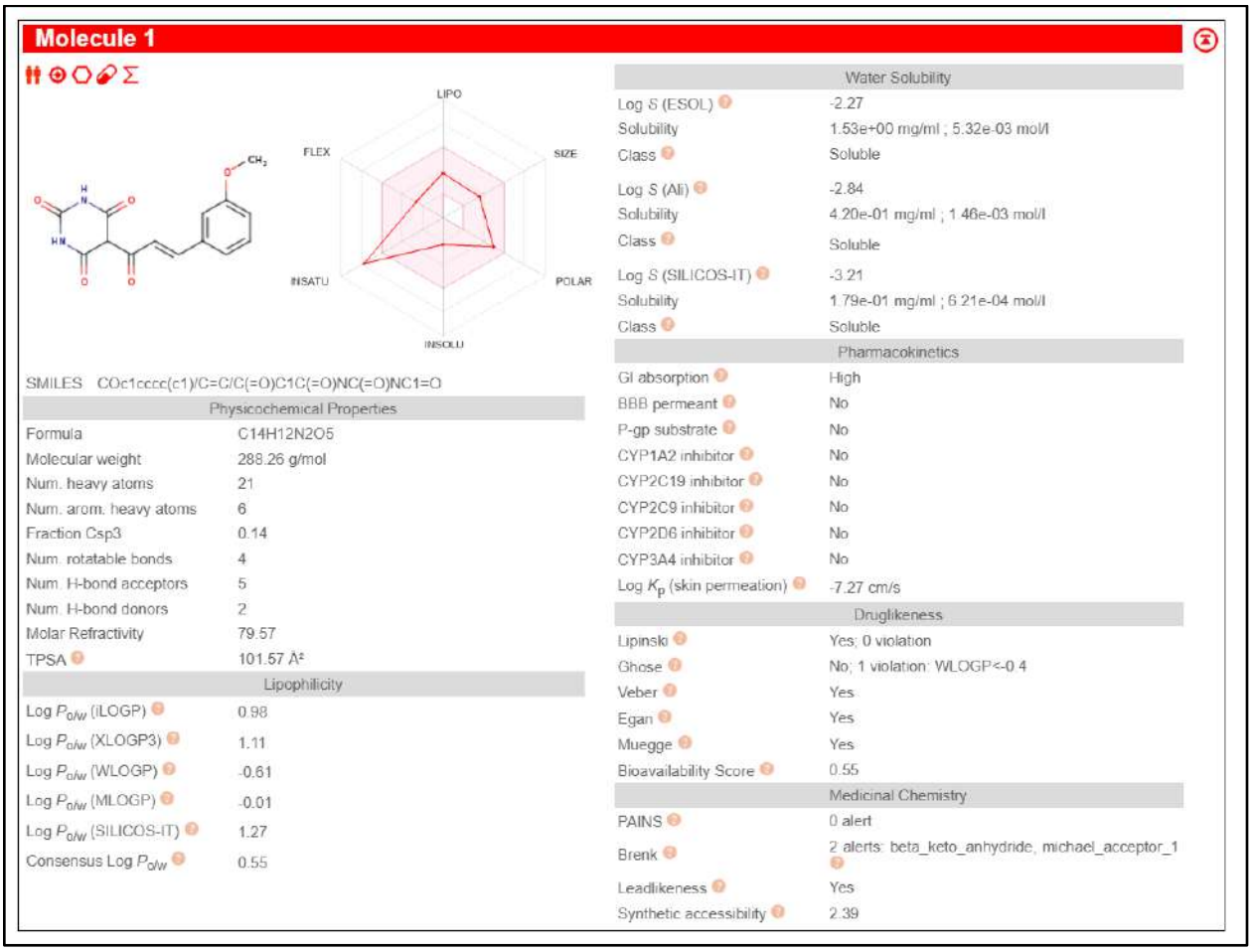

Figure 4: SwissADME profile of Compound ID (11129945)

Results and Discussion

A multi-step computational pipeline was employed in this study to identify and assess small-molecule inhibitors of *S. aureus* Fibronectin-binding proteins (FnBPs). The outcomes are presented in three phases: molecular docking of starting compounds, high-throughput virtual screening, and pharmacokinetic profiling.

Molecular Docking of Starting Compounds

A preliminary list of seven ligands—ML346 (CID 767276), Isovitexin (CID 162350), Gallic acid (CID 370), Quercetin (CID 5280343),

Triazolothiadiazole (CID 136376653), Morin (CID 5281670), and Benzoic acid (CID 82546561)—was docked against FnBPs (PDB ID: 2RKY). Both blind docking and focused docking methods were used.

As mentioned in Table 1. presents the binding energies of the ligands. Of these, Benzoic acid (CID 82546561) had the highest binding affinity with a fixed docking score of −8.61 kcal/mol. ML346 and Gallic acid came close with positive docking scores. Interestingly, in all the ligands, the focused docking binding energies were always less compared to blind docking, attesting to the existence of a defined, high-affinity binding pocket on FnBPs.

Table 1 Cluster table analysis of ligands (RSMD table)

Sr.	Source	Compound ID	Compound name	Blind dock (Kcal/mol)	Fix dock (Kcal/mol)
1	Pubchem	136376653	Triazolothiadiazole	-6.63	-6.77
2	Pubchem	767276	ML346	-6.04	-7.34
3	Pubchem	162350	Isovitexin	-6.00	-6.84
4	Pubchem	82546561	Benzoic acid	-5.84	-8.61
5	Pubchem	370	Gallic acid	-5.68	-7.29
6	Pubchem	5280343	Quercetin	-5.43	-5.92
7	Pubchem	5281670	Morin	-5.29	-6.05

Table 2 Fixed dock coordinates

Sr.	Compound name	Compound ID	X centre	Y centre	Z centre
1	Morin	5281670	16.694	5.831	20.654
2	Gallic acid	370	21.081	23.211	20.396
3	Benzoic acid	82546561	17.331	6.361	19.555
4	ML346	767276	17.273	7.579	19.562
5	Isovitexin	162350	11.098	7.509	22.194
6	Quercetin	5280343	17.256	6.295	20.123
7	Triazolothiadiazole	136376653	11.398	12.568	4.419

Visualization of the Docked Compound

Visualization of docked complexes gave detailed information on the molecular interactions between ligands and the FnBPs binding pocket. Of the screened compounds, ML346 exhibited favorable binding by establishing hydrogen bonds and hydrophobic interactions with important residues in the FnBP active site, such as Thr214, Trp213, and Glu44. In comparison to quercetin and morin, which exhibited weaker contacts and thus lower binding affinities, gallic acid exhibited additional hydrogen bonding and π–π

stacking interactions in line with its polyphenolic scaffold. This analysis emphasizes the effectiveness of gallic acid in stabilizing FnBP–ligand interactions, where its aromatic rings and hydroxyl substitutions played a major role in binding strength.

The binding pose and conformational stability of the top compound were further analyzed. The RMSD clustering histogram revealed a predominant cluster of conformations at the lowest binding energy, as shown in Figure. 5 suggesting a stable binding mode.

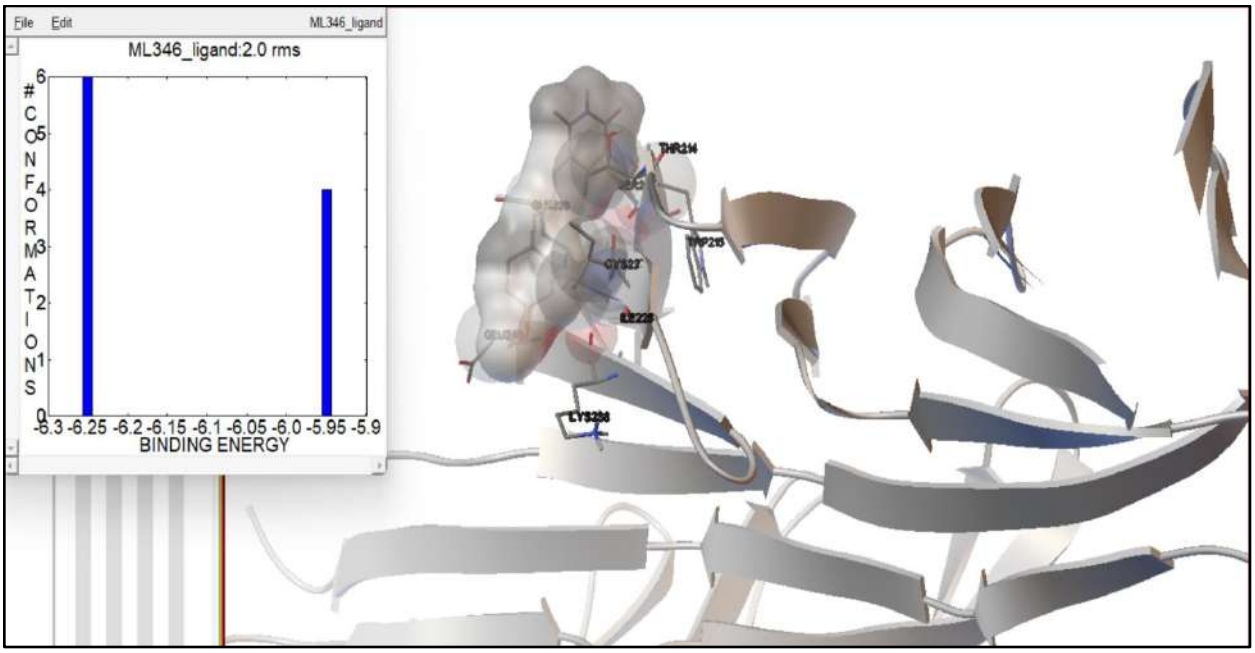

Figure 5. Visualization of ML346 complex via Autodock

Virtual Screening for Novel Inhibitors

Following the initial docking, high-throughput virtual screening was carried out using compound libraries structurally related to the seven

ligands. Each library contained certain compounds, which were filtered using Lipinski's Rule of Five. This reduced the pool of candidates substantially; for example, the Benzoic acid library was narrowed down to 29 compounds.

Herein, Table 3 presents the leading hits from the screening. A few analogs showed more favorable binding energies than the parent compounds. Benzoic acid analogs exceeded others in strength, as one of the analogs provided a fixed docking score of −8.73 kcal/mol, higher than the parent molecule.

Analogues of Quercetin and Gallic acid also exhibited increased binding affinities.

Table 3 Virtual Screening result via Autodock Vina (RSMD value) and Lipinski Rule of 5

Sr.	Compound name	Source	Compound ID	Blind value (Kcal/mol)	Fix value (Kcal/mol)	Before filter	After filter
1	Morin	PubChem	5281670	-7.1	-7.9	1000	18
2	Gallic acid	PubChem	370	-6.4	-6.6	984	31
3	Benzoic acid	PubChem	82546561	-6.5	-6.6	1000	29
4	ML346	PubChem	767276	-7.2	-6.5	185	22
5	Isovitexin	PubChem	162350	-6.8	-7.4	1000	31
6	Quercetin	PubChem	5280343	-7.1	-5.9	1000	53
7	Triazolothiadiazole	PubChem	136376653	-5.4	-4.0	1	1

In Silico Physicochemical and Pharmacokinetic Profiling

The best-scoring ligands underwent in silico ADMET profiling to check for their drug-likeness as well as pharmacokinetic properties. The most informative physicochemical descriptors are listed in Table 4. The chosen compounds exhibited molecular weights of between 261.23 g/mol and 288.26 g/mol, within the ideal range of oral drugs. Their calculated lipophilicity (ClogP) values varied between 0.20 and 0.87, suggesting an appropriate balance between hydrophilicity and lipophilicity. Importantly, all compounds adhered to Lipinski's Rule of Five, including acceptable ranges for hydrogen bond donors and acceptors, indicating favorable drug-like properties.

The predicted pharmacokinetic parameters are listed in Table 5. A key observation was that the high-affinity compounds exhibited high gastrointestinal (GI) absorption, highlighting their potential suitability for oral delivery. None of the compounds screened was anticipated to penetrate the blood–brain barrier (BBB), a positive safety characteristic since it reduces the likelihood of central nervous system side effects. Although several of the compounds were found to be non-substrate for P-glycoprotein (P-gp), thus evading efflux-related constraints, their uniform oral bioavailability scores of 0.55 indicate encouraging pharmacokinetic properties.

Table 4 SwissADME physiochemical parameters

Compound ID	Consensus Log P	MW (150-500)	HBD (0-5)	HBA (0-10)	Rotatable bonds (<10)	TPSA (60-140 A)	Ali Solubility (mg/ml)
11129945	0.55	288.26	2	5	4	101.57	1.53E+00
71267590	0.2	261.23	2	5	2	101.73	4.19E+00
5115520	0.87	270.24	2	4	1	101.57	1.68E+00
2246125	0.69	276.24	2	5	3	93.73	1.98E+00
15293760	2.08	302.24	5	6	1	111.13	3.04E-02
5280445	1.73	302.24	5	6	1	111.13	5.63E-02
5280666	2.18	286.24	3	6	2	100.13	2.61E-02
5318214	2.13	302.24	5	6	2	100.13	3.70E-02
6477685	2.5	206.19	1	6	3	96.97	2.20E-02
11255393	2.12	180.16	2	6	2	100.13	3.06E-02

This study directly responds to the paucity of systematic computer-based studies on FnBPs inhibition in *S. aureus*. Through a systematic *in silico* workflow, this research identified several new compounds

with high potential as anti-virulence compounds. Inhibition of a virulence factor like FnBPs, which plays a crucial role in bacterial adhesion, immune evasion, and biofilm development, provides an alternative strategy for traditional antibiotic treatment. Blocking FnBPs can reduce the virulence of *S. aureus* defenses, thus minimize colonization and increase immune clearance at the cost of decreasing the selective pressure driving antibiotic resistance.

One particularly notable result was that fixed docking consistently yielded better binding energies than blind docking, further supporting the existence of a clear high-affinity binding site within FnBPs. This increases confidence that the identified site is a genuine inhibitory pocket to be used therapeutically.

Among the screened compounds, Benzoic Acid (−8.61 kcal/mol), ML346 (−7.34 kcal/mol), and Gallic Acid (−7.29 kcal/mol) proved to be the best inhibitors, with strong affinities and good pharmacokinetic predictions. These findings are in line with past evidence that flavonoids and small aromatic acids have diverse biological activities, thus confirming their therapeutic potential. The *in silico* ADMET profiling further enhanced the relevance of these compounds, with good GI absorption, no BBB permeability, and drug-likeness filter compliance. Although some of these compounds could be subject to efflux transport limitations, this does not rule out their development, especially in combination therapies using standard antibiotics.In all, the study proves the successful identification of highly effective FnBPs inhibitors, such

as Benzoic Acid, ML346, and Gallic Acid, which are a sound foundation for the creation of new anti-adhesion and anti-virulence treatments against antibiotic-resistant *S. aureus.*

Conclusion

This research delivers a thorough *in silico* inhibition screening for the Fibronectin-binding proteins (FnBPs) of *S. aureus*, an important virulence factor implicated in adhesion, biofilm development, and immune evasion. Its findings demonstrate the promise of an anti-virulence therapeutic strategy, which diverges from the conventional bactericidal strategy a strategy that seeks to incapacitate the pathogen instead of killing it. This is a promising avenue in drug discovery to counteract antimicrobial resistance.

By a well-ordered computational workflow encompassing molecular docking, virtual screening, and ADMET profiling, various small-molecule ligands were screened with high predicted affinities to bind with FnBPs. Significantly, Benzoic Acid (−8.61 kcal/mol), ML346 (−7.34 kcal/mol), and Gallic Acid (−7.29 kcal/mol) were found to be lead candidates possessing good drug-like properties and strong prospects as anti-adhesion drugs. These results support the validity of targeting FnBPs as a drug development strategy, with compounds that potentially could inhibit colonization and virulence without applying the selective pressure typically wielded by traditional antibiotics.

Experimental verification of these computational predictions should be the focus of future studies. This

entails in vitro biochemical assays to establish direct inhibition of FnBPs, and subsequent in vivo infection models to determine their effect on bacterial adhesion, biofilm formation, and pathogenicity. The possibility of synergy between FnBPs inhibitors and traditional antibiotics should also be investigated to determine if they could be used in combination therapy against multidrug-resistant *S. aureus*.

Generally, this study provides a sound basis for the discovery of new anti-virulence drugs acting against FnBPs. By moving from predictive computational studies to testing in laboratory and clinical settings, these studies could lead to the design of next-generation therapeutics that will help block antibiotic resistance and enhance infection outcomes in *S. aureus* infections.

Table 5 Pharmacokinetic Properties.

Compound ID	Formula	GI absorption	BBB permeant	Pgp substrate	Log kp (cm/s) Skin permeation	Bioavailability	Synthetic Accessibility
11129945	C14H12N2O5	High	No	No	-7.27	0.55	2.39
71267590	C12H11N3O4	High	No	No	-7.27	0.55	2.32
5115520	C14H10N2O4	High	No	No	-7.34	0.55	3.01
2246125	C13H12N2O5	High	No	No	-7.34	0.55	2.44
15293760	C16H12O6	High	No	No	-6.08	0.55	3.13
5280445	C15H10O6	High	No	No	-6.25	0.55	3.02
5280666	C16H12O6	High	No	No	-5.93	0.55	3.06
5318214	C16H12O6	High	No	No	-6.1	0.55	3.1
6477685	C17H12O6	High	No	No	-5.88	0.55	3.14
11255393	C16H12O6	High	No	No	-6.01	0.55	3.12

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