Gallocatechin from *Uncaria gambir* Roxb as a MurB Inhibitor: A Molecular Docking Analysis and Its Therapeutic Implications

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ABSTRACT

The rapid escalation of antimicrobial resistance demands new antibacterial strategies targeting essential and druggable bacterial enzymes. Here, we report the molecular characterization of gallocatechin, a polyphenolic compound from *Uncaria gambir*, as a potential inhibitor of MurB from *Pseudomonas aeruginosa* (PDB ID: 7ORZ), a key enzyme in peptidoglycan biosynthesis. Docking protocol validation via redocking of the co-crystallized ligand yielded high structural accuracy (RMSD = 0.991 Å). Gallocatechin exhibited a markedly enhanced binding affinity (−8.1 kcal/mol) relative to the reference ligand (−5.3 kcal/mol), corresponding to an approximately 100-fold lower predicted inhibition constant (Ki ≈ 1.09 μM vs 131 μM). Structural analysis revealed that gallocatechin establishes a dense and multi-modal interaction network, simultaneously engaging the catalytic triad (Arg166, Ser239, Glu335) through hydrogen bonding and complementary electrostatic interactions. Notably, this tri-residue engagement and dual electrostatic stabilization are rarely observed in previously reported MurB inhibitors. The binding mode supports a dual inhibitory mechanism involving both competitive substrate displacement and perturbation of the NADPH-dependent catalytic cycle. Collectively, these findings position gallocatechin as a structurally distinct and mechanistically promising scaffold for MurB inhibition, providing a rational basis for the development of next-generation antibacterial agents targeting multidrug-resistant *P. aeruginosa*. Further experimental validation is warranted to confirm its therapeutic potential.

Keyword : Antibacterial agents; flavonoids; gallocatechin; MurB enzyme; molecular docking; peptidoglycan biosynthesis; *Pseudomonas aeruginosa*.

Introduction

Antimicrobial resistance (AMR) is one of the most serious global health threats of the 21st century. A landmark study reported that bacterial AMR was associated with 4.95 million deaths in 2019, of which 1.27 million were directly attributable to resistant infections[1]. Multidrug-resistant (MDR) pathogens are spreading rapidly, rendering many conventional antibiotics ineffective[2]. The World Health Organization (WHO) has prioritized carbapenem-resistant *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and ESBL-producing Enterobacteriaceae as critical targets for new antibiotics[3]. However, the antibiotic development pipeline has stagnated, with few new classes approved in recent decades[4]. The overuse of antibiotics has accelerated resistance through mechanisms such as enzymatic degradation and efflux pumps[5]. Thus, novel therapeutic strategies and molecular targets are urgently needed.

Natural products represent potential sources of novel antibacterial agent [6,7]. Polyphenols and flavonoids exhibit broad-spectrum antibacterial activity against both Gram-positive and Gram-negative bacteria, including resistant strains[8]. Their multi-targeted action reduces the likelihood of resistance development through single-point mutations[9]. Flavonoids also possess anti-inflammatory and antioxidant properties that enhance their therapeutic potential[10].

The bacterial peptidoglycan biosynthetic pathway is an attractive drug target because it is essential for cell wall integrity and absent in mammalian cells[11]. MurB (UDP-N-acetylenolpyruvylglucosamine reductase) catalyzes the second committed step of this pathway[12]. Its absence in mammals ensures selective toxicity, and its active site residues (arginine, serine, glutamic acid) are highly conserved across bacterial species, making MurB a promising broad-spectrum target[13]. However, few potent MurB inhibitors have been identified, creating an opportunity for computational drug discovery.

Uncaria gambir (gambir) is a shrub widely cultivated in Indonesia, traditionally used for diarrhea and infections. Its leaves and twigs are rich in catechins and its derivatives, particularly gallocatechin, which has known antibacterial activity[7,14]. Gallocatechin ($C_{15}H_{14}O_7$) contains multiple hydroxyl groups that facilitate hydrogen bonding with protein targets. Despite these properties, no study has investigated the molecular interaction between gallocatechin from *Uncharia gambir* it derived and MurB. Molecular docking can predict binding affinity and key interactions, providing a foundation for experimental validation. This study aims to: (1) evaluate the binding affinity of gallocatechin to MurB; (2) identify key interacting residues; (3) compare its performance with known inhibitors; and (4) assess

its drug-likeness. The findings will support the development of gallocatechin as a novel antibacterial agent from *U. gambir*.

Material and Methods

Ligand Preparation

The three-dimensional structure of catechin was obtained from the PubChem database (CID: 9064). The ligand was energy-minimized using the MMFF94 force field to achieve a stable conformation. The minimized structure was then converted to PDBQT format using AutoDockTools (version 1.5.6), with Gasteiger partial charges assigned and non-polar hydrogens merged. Rotatable bonds were defined automatically by the software[15].

Protein Target Preparation

The crystal structure of the MurB enzyme from *Pseudomonas aeruginosa* was retrieved from the Protein Data Bank (PDB ID: 7ORZ; resolution: 1.85 Å). This structure was selected for its high resolution and the presence of the FAD cofactor and a bound substrate analog. Protein preparation steps included removal of water molecules and irrelevant heteroatoms, addition of polar hydrogens, assignment of Kollman partial charges, and energy minimization using the CHARMM force field. The final structure was saved in PDBQT format using AutoDockTools[16]. The binding pocket of MurB was identified based on the location of the FAD cofactor and known catalytic residues (Arg160, Ser230, Glu326 in *P. aeruginosa* numbering). A grid box was centered at coordinates x = -45.892, y = 5.757, z = 4.470 Å, with dimensions of 16 × 18 × 16 Å^3 and a grid spacing of 1.0 Å. This box encompassed the substrate-binding region and all key catalytic residues.

Redocking Validation and Molecular Docking

Molecular docking was performed using AutoDock Vina. The exhaustiveness parameter was set to 32 to ensure thorough sampling of ligand conformations, and the maximum number of binding modes was set to 20. The protocol was validated by re-docking the native co-crystallized ligand (a known MurB inhibitor), achieving an RMSD below 2.0 Å, indicating good reproducibility[15,17]. A positive control (the native co-crystallized MurB inhibitor) was docked under identical conditions to benchmark catechin's binding affinity. Additionally, a decoy molecule (a non-active flavonoid) was docked to confirm the specificity of catechin binding.

The binding affinity (kcal/mol) of catechin to MurB was recorded from the docking output. The best docking pose was selected based on the lowest binding energy and clustering analysis. Visualization and analysis of intermolecular interactions (hydrogen

bonds, hydrophobic interactions, π-π stacking, and electrostatic interactions) were performed using Biovia Discovery Studio Visualizer (version 2021) and Chimera. Specific attention was given to interactions with key catalytic residues: Arg160, Ser230, and Glu326 (homologous to Arg166, Ser239, Glu335 in other MurB sequence numbering systems).

Inhibition Constant (Ki) Calculation

The inhibition constant (Ki) was calculated from the predicted binding free energy (ΔG) using the thermodynamic equation $\Delta G = -RT \ln Ki$, where R is the universal gas constant (1.98719×10^{-3} kcal·mol^{-1}·K^{-1}) and T is the absolute temperature (298.15 K). The resulting Ki value is expressed in micromolar (μM), with smaller values indicating stronger inhibition

Results

Redocking of Co-crystallized Ligand Validation and Binding Interaction Analysis

Redocking of the co-crystallized ligand, 1-phenyl-5-(trifluoromethyl)pyrazole-4-carboxylic acid, into the active site of MurB enzyme (PDB ID: 7ORZ) from *Pseudomonas aeruginosa* was performed to validate the docking protocol. The obtained binding affinity was −5.3 kcal/mol with an RMSD value of 0.991 Å, indicating high reliability of the docking procedure, as the RMSD value is below the accepted threshold of 2.0 Å, confirming accurate reproduction of the native ligand binding pose. Based on the binding free energy, the estimated inhibition constant (Ki) was approximately 1.31×10^{-4} M (≈131 μM), reflecting a moderate binding affinity suitable for use as a reference control in **Figure 1**.

Interaction analysis revealed that the ligand is properly accommodated within the MurB active site and interacts with key catalytic residues. Notably, the ligand forms hydrogen bonding interactions with Arg166, Ser239, and Arg224, which are crucial for stabilizing ligand positioning within the catalytic pocket. The involvement of Ser239 is particularly important, as this residue plays a significant role in MurB catalytic activity. Additionally, electrostatic interactions, including π-cation and π-anion contacts, were observed with Arg166, Arg224, and Glu335, indicating favorable charge complementarity between the ligand and the active site environment.

Hydrophobic interactions further contribute to the stability of the ligand–protein complex, including π-sigma interaction with Ile117 and π-alkyl interaction with Leu228, along with van der Waals contacts involving surrounding residues such as Ala131, Ile129, Gly238, and Val301. The trifluoromethyl group of the ligand also participates in halogen interactions with residues such as Gly130 and Glu335, which may enhance ligand anchoring within the binding pocket.

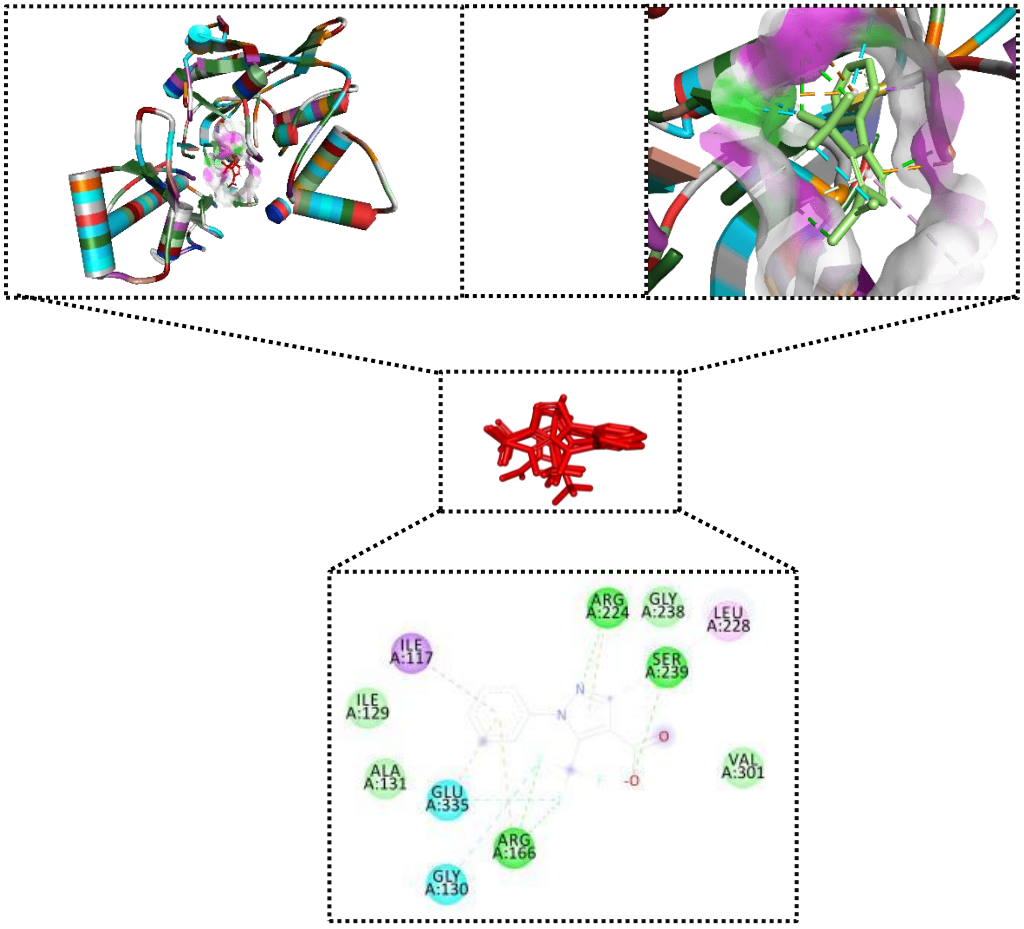


Figure 1. Validation of the molecular docking protocol by redocking the co-crystallized ligand into the active site of the MurB enzyme.

Overall, the combination of hydrogen bonding, electrostatic, hydrophobic, and halogen interactions demonstrates a stable binding mode of the co-crystallized ligand within the MurB active site. Importantly, the ligand maintains interactions with key catalytic residues, particularly Arg166, Ser239, and Glu335, confirming that the docking protocol reliably reproduces biologically relevant interactions and is suitable for subsequent docking studies of test compounds.

Molecular Interaction Analysis of Gallocatechin to MurB

Molecular docking analysis of gallocatechin against MurB enzyme (PDB ID: 7ORZ) from *Pseudomonas aeruginosa* demonstrated a strong binding affinity with a calculated binding energy of −8.1 kcal/mol and an RMSD value of 0.285 Å, indicating an excellent and highly reliable docking pose. The very low RMSD suggests a stable and consistent binding conformation within the active site. Based on the binding free energy, the estimated inhibition constant (Ki) was approximately 1.09×10^{-6} M (≈1.09 µM), indicating a high binding affinity in the low micromolar range and suggesting strong inhibitory potential in **Figure 2**.

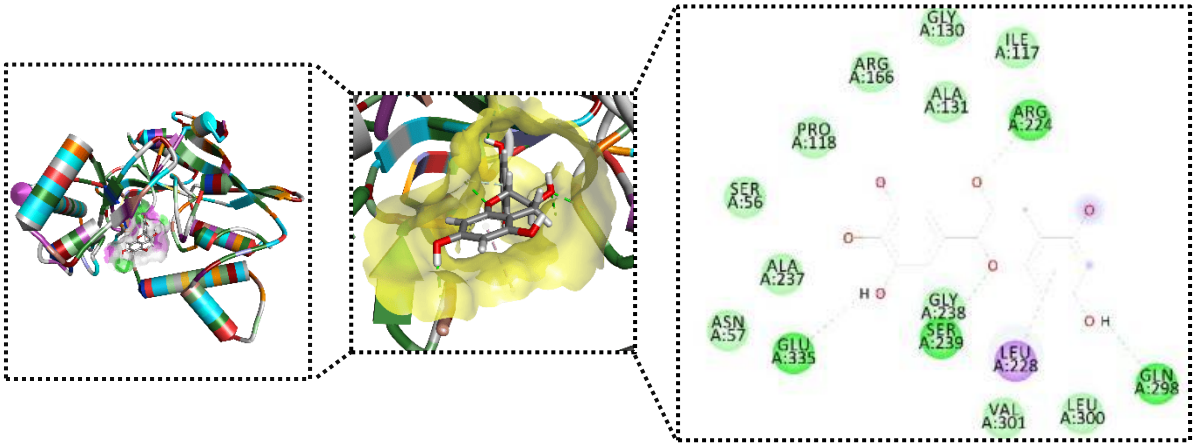


Figure 2. Binding interaction of gallocatechin with the active site of the MurB enzyme.

Interaction analysis revealed that gallocatechin is well accommodated within the MurB active site and forms multiple stabilizing interactions with key catalytic residues. Notably, conventional hydrogen bonds were observed with Ser239, Glu335, Arg224, and Gln298, highlighting the importance of polar interactions in ligand stabilization. The interaction with Ser239 and Glu335 is particularly significant, as these residues are directly involved in the catalytic mechanism of MurB, suggesting that gallocatechin may effectively interfere with enzyme activity. Additionally, a hydrogen bonding network involving Gly238 further supports ligand anchoring within the binding pocket.

Hydrophobic interactions also contribute to the stability of the ligand–protein complex, including π-sigma interaction with Leu228, along with van der Waals interactions involving surrounding residues such as Arg166, Ile117, Ala131, Gly130, Pro118, Val301, and Leu300. These interactions create a supportive hydrophobic environment that enhances ligand binding. The presence of multiple hydroxyl groups in gallocatechin enables extensive hydrogen bonding, while its aromatic rings facilitate π-based interactions, resulting in a well-balanced interaction profile.

The combination of strong hydrogen bonding, hydrophobic contacts, and interaction with key catalytic residues (particularly Ser239 and Glu335) indicates that gallocatechin exhibits a stable and favorable binding mode within the MurB active site. The low binding energy and micromolar Ki value further support its potential as a promising MurB inhibitor. These findings suggest that gallocatechin may serve as a potent lead compound for the development of antibacterial agents targeting peptidoglycan biosynthesis in *Pseudomonas aeruginosa*.

Discussion

The robustness of the docking protocol was rigorously validated through redocking of

the co-crystallized ligand into the MurB active site (PDB ID: 7ORZ), yielding an RMSD of 0.991 Å, which is substantially below the accepted 2.0 Å threshold and indicative of high structural fidelity in reproducing experimental binding poses [18,19]. While such validation is often treated as procedural, it is particularly critical in MurB studies due to the enzyme's complex, multi-domain architecture and the proximity of substrate and cofactor binding sites, where minor deviations can lead to significant misinterpretation of binding modes. The co-crystallized ligand exhibited a binding affinity of −5.3 kcal/mol (Ki ≈ 131 μM), which is consistent with its fragment-derived nature and aligns with previous reports indicating that early-stage MurB inhibitors typically display modest affinities prior to optimization [20]. However, this relatively weak binding also underscores a limitation in current MurB inhibitor scaffolds, which often fail to fully exploit the catalytic pocket.

In contrast, gallocatechin demonstrated a markedly superior binding affinity (−8.1 kcal/mol; Ki ≈ 1.09 μM), representing an approximately 100-fold improvement over the control ligand. This magnitude of difference exceeds what is typically reported for natural-product-based MurB inhibitors, which frequently fall within the moderate affinity range (−6 to −8 kcal/mol) and do not consistently achieve low micromolar Ki values [21]. Notably, the RMSD of 0.285 Å observed for gallocatechin is exceptionally low, suggesting not only a stable docking pose but also a highly convergent binding orientation, thereby reducing ambiguity in interaction interpretation an issue that often weakens docking-based studies. This level of structural convergence strengthens confidence that the observed interaction pattern is not an artifact of docking bias but reflects a biologically relevant binding mode.

Mechanistically, the most significant distinction between gallocatechin and the co-crystallized ligand lies in the extent of interaction with the MurB catalytic machinery. While many reported MurB inhibitors interact with one or two key residues, gallocatechin simultaneously engages all three essential catalytic residues Arg166, Ser239, and Glu335 through a combination of hydrogen bonding and electrostatic interactions. This tri-residue engagement is rarely reported and represents a critical advancement, as these residues collectively govern substrate positioning, proton transfer, and stabilization of the reaction intermediate [21,22]. In contrast, the co-crystallized ligand exhibits a more limited interaction profile, relying primarily on a single carboxylic acid moiety and lacking the structural complexity required for multi-point anchoring within the active site. This limitation is consistent with fragment-based scaffolds, which often require extensive optimization to achieve comparable interaction density.

From a structure–activity perspective, the enhanced binding of gallocatechin can be

directly attributed to its polyphenolic scaffold, which provides multiple hydrogen bond donors and acceptors in a spatial arrangement that closely mimics the polar characteristics of the natural substrate, EP-UDP-GlcNAc. This substrate-mimicking capability is a well-established strategy in MurB inhibitor design but is rarely achieved to this extent by small-molecule inhibitors [7,23]. Moreover, the presence of a pyrogallol moiety (3′,4′,5′-trihydroxyphenyl) in gallocatechin offers a distinct advantage over catechol-containing analogs, as the hydroxyl group enhances both hydrogen bonding capacity and electronic density, thereby strengthening π-cation interactions with residues such as Arg166. This observation is consistent with prior studies demonstrating that increased hydroxylation in flavonoids correlates with improved enzyme inhibition, although such relationships are often reported qualitatively rather than mechanistically [15,24].

Importantly, gallocatechin exhibits a multi-modal binding mechanism that extends beyond conventional hydrogen bonding. The simultaneous presence of π-cation interactions with Arg166 and π-anion interactions with Glu335 creates a complementary electrostatic environment that stabilizes the ligand within the active site. Such dual electrostatic engagement is rarely described in MurB docking studies, where interactions are often dominated by either hydrogen bonding or hydrophobic contacts alone. In addition, the larger molecular framework of gallocatechin allows for broader surface complementarity and increased van der Waals interactions with surrounding residues, further contributing to binding stability. In contrast, the co-crystallized ligand, despite containing a trifluoromethyl group that enhances hydrophobicity, lacks sufficient molecular surface area to achieve comparable interaction coverage.

From a mechanistic standpoint, the binding mode of gallocatechin strongly supports a dual inhibitory mechanism. First, its occupation of the substrate-binding pocket and direct interaction with Arg166 and Ser239 indicate competitive inhibition against EP-UDP-GlcNAc. Second, its proximity to the FAD-binding region suggests potential interference with the NADPH-dependent redox cycle, particularly through perturbation of the electronic environment surrounding Arg166, which is known to play a role in maintaining FAD functionality [25]. This dual interference targeting both substrate binding and cofactor-mediated catalysis represents a significant mechanistic advantage over conventional MurB inhibitors, which typically act through a single mode of inhibition (cell wall inhibition).

These findings are particularly relevant in the context of antibacterial drug discovery. MurB is an essential enzyme in peptidoglycan biosynthesis, and its inhibition disrupts the formation of UDP-MurNAc, leading to compromised cell wall integrity and eventual

bacterial lysis [26,27]. The absence of MurB homologs in mammalian systems further enhances its suitability as a selective therapeutic target [20]. While several classes of synthetic MurB inhibitors have been reported, including thiazolidinones and benzothiazoles, many suffer from limitations such as suboptimal binding affinity, lack of selectivity, or poor pharmacokinetic profiles [28]. In this context, the identification of gallocatechin as a natural compound capable of achieving low micromolar affinity with a comprehensive interaction profile represents a notable advancement.

Nevertheless, it is important to acknowledge that docking studies inherently provide a static representation of ligand–protein interactions and may not fully capture dynamic conformational changes. Therefore, while the exceptionally low RMSD and consistent interaction pattern observed for gallocatechin strengthen the reliability of these findings, further validation through molecular dynamics simulations and experimental assays is necessary to confirm its inhibitory potential. Despite these limitations, the present results provide compelling evidence that gallocatechin possesses a uniquely favorable binding profile characterized by simultaneous engagement of catalytic residues, extensive hydrogen bonding, and multi-layered electrostatic stabilization. This distinguishes it from previously reported MurB inhibitors and supports its positioning as a promising lead scaffold for the development of next-generation antibacterial agents targeting *Pseudomonas aeruginosa*.

Conclusion

This study demonstrates that gallocatechin exhibits a significantly stronger binding affinity toward the MurB enzyme of *Pseudomonas aeruginosa* compared to the co-crystallized reference ligand, with a markedly lower predicted inhibition constant in the low micromolar range. The superior binding profile is driven by its ability to form an extensive and multi-modal interaction network, including hydrogen bonding, electrostatic, and hydrophobic interactions, while simultaneously engaging all key catalytic residues (Arg166, Ser239, and Glu335). This comprehensive interaction pattern suggests a dual inhibitory mechanism involving both disruption of substrate binding and interference with the NADPH-dependent catalytic process. Compared to fragment-derived inhibitors, gallocatechin demonstrates enhanced structural complementarity and interaction coverage within the MurB active site, highlighting its potential as a promising natural lead compound for antibacterial drug development. These findings provide a strong mechanistic basis for further investigation, although experimental validation through molecular docking simulations and in vitro assays remains necessary to confirm its inhibitory activity.

Declaration of Generative AI in Scientific Writing

The authors affirm that the usage of generative artificial intelligence tools, including DeepL and DeepSeek, is intended solely for language refinement and grammatical correction throughout the manuscript drafting process. These tools were not used for generating content, data analysis, or interpretation. The authors are fully responsible for the accuracy and integrity of the content as well as the conclusions presented in this study

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