MSW MANAGEMENT -Multidisciplinary, Scientific Work and Management Journal
ISSN: 1053-7899
Vol. 36 Issue 1, 2026, Pages 6258-6262

Evaluation of antibacterial activity of 3-formylchromone against *Acinetobacter baumannii* with molecular docking insights on a biofilm-associated proteins

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ABSTRACT
Acinetobacter baumannii (*A. baumannii*) is a multidrug-resistant (MDR) opportunistic pathogen associated with severe nosocomial infections and robust biofilm formation, necessitating the development of novel therapeutic strategies. In the present study, the antibacterial potential of 3-formylchromone (3-FC) was evaluated against a clinical isolate of *A. baumannii*, along with *in-silico* analysis targeting biofilm-associated proteins. Antibiotic susceptibility testing revealed a multidrug-resistant profile, with sensitivity observed only to tetracycline. The antibacterial activity of 3-FC was assessed using agar well diffusion, which demonstrated a zone of inhibition of 21 mm. The minimum inhibitory concentration (MIC) of 3-FC was determined to be 0.312 mg/mL using a broth microdilution method. At sub-MIC levels, 3-FC did not significantly affect bacterial metabolic activity, as confirmed by the Alamar Blue assay. Molecular docking studies revealed favorable binding interactions of 3-FC with key biofilm-associated proteins, including *OmpA* and *BfmR*. These findings suggest that 3-FC exhibits promising antibacterial and may be a potential antibiofilm activity against MDR *A. baumannii*. Further studies are required to validate its mechanism of action and explore its therapeutic applicability.

Keywords: *A. baumannii,* 3-formylchromone, antibacterial, docking

1. INTRODUCTION

Acinetobacter baumannii is a Gram-negative, aerobic, opportunistic coccobacillus that is oxidase-negative, catalase-positive, and non-fermentative. The World Health Organization (WHO) has classified it as a Group 1 priority pathogen because of its important role in hospital-acquired infections, particularly in patients with impaired immune systems. This prominence is largely attributed to its rapid acquisition of antimicrobial resistance and its exceptional capacity to develop biofilms on both biotic and abiotic surfaces (1).

Infections caused by *A. baumannii* commonly arise through contact with moist body sites such as mucosal membranes or damaged skin, leading to conditions including respiratory tract infections, bacteremia, and sepsis (2,3). Epidemiological observations suggest a higher prevalence of these infections in males, potentially linked to factors such as aging, lifestyle habits like smoking and alcohol use, and comorbidities including diabetes, renal diseases, and chronic obstructive pulmonary disease. The pathogenic success and colonization efficiency of *A. baumannii* are driven by multiple virulence determinants that facilitate adhesion, cytotoxicity, immune evasion, microbial competition, and genetic adaptability, with biofilm formation playing a central role (4). Important virulence factors include extracellular exopolysaccharides (EPS), the *BfmS/BfmR* two-component regulatory system, biofilm-associated protein (*bap*), the chaperone–usher pilus assembly system (*CsuA/B*), outer membrane protein A (*OmpA*), β-lactamase PER-1, and quorum sensing (QS) mechanisms (5–7). Together, these components contribute to critical infection processes such as host immune modulation, tissue damage, serum resistance, and adherence to epithelial surfaces. Notably, *bap* is essential for bacterial aggregation, intercellular adhesion, and the establishment of robust biofilms (6).

Through signaling molecules known as autoinducers (AIs), QS is a bacterial communication system that allows microorganisms to sense population density. This allows them to coordinate gene expression and adapt to changes in their environment. Because QS is necessary for the formation of biofilms and multidrug resistance (MDR), disrupting QS with quorum sensing inhibitors or quorum quenchers has emerged as a successful antibacterial strategy (8). QS systems are widely distributed among pathogenic bacteria, including *Escherichia coli*, *Vibrio cholerae*, *Acinetobacter baumannii*, and *Pseudomonas aeruginosa*, as well as plant pathogens such as *Erwinia* and *Ralstonia*. Biofilm formation is a multifactorial process involving cellular aggregation, adhesion, pilus expression, iron acquisition, and virulence factor secretion (9,10). The exopolysaccharide (EPS) matrix provides structural stability and acts as a barrier against antibiotics, contributing to enhanced resistance (11).

In *A. baumannii*, genes such as *plcN*, *lasB*, and *blaPER-1*, along with the *BfmRS* regulatory system, are associated with virulence, biofilm development, and antibiotic resistance. Biofilm-associated bacteria exhibit reduced susceptibility to antibiotics like gentamicin, piperacillin, and ceftazidime due to factors such as limited drug penetration, metabolic heterogeneity, and persistence of dormant cells(12).

With the rapid rise of antimicrobial resistance (AMR), targeting quorum sensing (QS) using natural compounds has emerged as a promising strategy for developing novel anti-infective therapies (13). Compounds like rosmarinic acid, quercetin, catechin, and apigenin have demonstrated significant antibiofilm and anti-QS activities, drawing attention to naturally occurring chromone derivatives for their diverse biological potential (14).

3-Formylchromone (3-FC), a member of the chromone family, is an oxygen-containing heterocyclic compound characterized by a formyl (-CHO) group at the third position of the chromone ring. Its molecular formula ($C_{10}H_6O_3$) and conjugated aromatic structure contribute to its stability and a variety of biological processes. 3-FC has antibacterial, anti-inflammatory, and antioxidant, properties, making it a promising lead compound in drug discovery (15–17). Moreover, its reactive aldehyde functionality enables its use in the synthesis of pharmaceuticals and other industrial products (16).

Here, we understand the antibacterial potential of 3-FC against *A. baumannii*, an opportunistic MDR pathogen. Notably, the antimicrobial activity of 3-FC against *A. baumannii* remains largely unknown. Therefore, this research attempts to close this gap by assessing its antibacterial efficacy, complemented by *in-silico* analyses to elucidate its possible mechanisms of action, particularly targeting biofilm-associated pathways.

2. Methodology

2.1 Bacterial Strain and Culture Conditions: The *A. baumannii* clinical isolate used in this study was obtained from archived cultures in the clinical microbiology repository of Saveetha Dental College and Hospitals, Chennai, India. Its identity was confirmed using the VITEK 2 system (18). The isolate was routinely cultured aerobically in Luria-Bertani (LB) broth (HiMedia, India) at 37 °C for 24 h under continuous shaking at 100 rpm.

2.2 3-Formylchromone (3-FC) : 3-Formylchromone (3-FC) is a naturally occurring bioactive compound known for its diverse biological characteristics, such as antioxidant, antibacterial, anti-inflammatory, and anticancer capabilities. According to the manufacturer's recommendations, 3-FC was purchased from Sigma-Aldrich (USA) for the current investigation and kept at room temperature.

2.3 Antibiotic Susceptibility Testing: According to the criteria of the Clinical and Laboratory Standards Institute (CLSI), the Kirby-Bauer disc diffusion method was used to assess the antibiotic susceptibility of the clinical isolate of *A. baumannii* (19). Briefly, bacterial inoculum of *A. baumannii* were prepared and uniformly spread using swabs moistened with suspension (bacteria) on Mueller-Hinton agar (MHA) plates. Standard antibiotic discs with the following concentrations, Piperacillin/Tazobactam (100/10 µg), Tetracycline (30 µg), Imipenem (10 µg), Ceftriaxone (30 µg), Cefotaxime (30 µg), Cefixime (5 µg), and Meropenem (10 µg) were placed on the agar surface to assess the susceptibility of the isolate to conventional antibiotics.

2.4 Antimicrobial testing of 3-Formylchromone (3-FC): The antibacterial activity of 3-FC was assessed against *A. baumannii* clinical isolate using the agar well diffusion method (20). A sterile cork borer was used to evenly swab the bacterial suspension onto MHA plates (HiMedia, Mumbai, India), and 8 mm wells were aseptically bored. 40 µL of 3-FC (40 mg/mL) was added to each well; the positive control was a common antibiotic, while the negative control was DMSO. Following 24 hr incubation at 37°C, zone of inhibition (ZOI) diameter was used to determine the antibacterial activity.

2.5 Evaluation of MIC in and cell viability in A. baumannii: Two-fold microdilution method was applied to understand the minimum inhibitory concentration (MIC) of 3-FC against *A. baumannii* (21). The assay was performed using serial concentrations of 3-FC ranging from 10 mg/mL to 0.019 mg/mL, in both the presence and absence of the test conditions. After incubation, 2,3,5-triphenyl tetrazolium chloride (TTC) was added as a viability indicator. The MIC was defined as the lowest concentration of 3-FC that completely inhibited visible bacterial growth. The effect of 3-FC on the metabolic activity of *A. baumannii* at the sub-MIC level was evaluated using the Alamar Blue test, a previously developed method (22). *A. baumannii* were grown in LB broth for 24 h at 37 °C, both with and without a sub-MIC dosage of 3-FC (0.156 mg/mL). Following incubation, the Alamar Blue reagent before being left to continue incubating in the dark. At 560 nm for excitation and 590 nm for emission, fluorescence was seen.

MSW MANAGEMENT -Multidisciplinary, Scientific Work and Management Journal
ISSN: 1053-7899
Vol. 36 Issue 1, 2026, Pages 6258-6262

2.8 Molecular docking of 3-FC: 3-Formylchromone (3-FC; PubChem CID: 87112), a chromone derivative with a molecular weight of 174.15 g/mol comprising 10 carbon, 6 hydrogen, and 3 oxygen atoms ($C_{10}H_6O_3$), was subjected to an *in silico* molecular docking study. Its chemical structure was retrieved and verified from the PubChem database (NCBI, NIH).

2.9 Structure-based docking and binding interaction analysis: Due to their involvement in biofilm formation and pathogenicity, two crystal structures from *A. baumannii* were chosen as molecular targets to the compound 3-FC: the *OmpA* peptidoglycan-binding domain (PDB ID: 4G4Y) and the N-terminal domain of *BfmR* (PDB ID: 5HM6). Three-dimensional structures were taken from the RCSB Protein Data Bank and produced using BIOVIA Discovery Studio Visualizer 2024 (v24.1.0.23298; Dassault Systèmes Biovia Corp.) by removing superfluous chains, water molecules, and co-crystallized ligands. Next came the addition of Gasteiger charges and polar hydrogens.

3.0 STATISTICAL ANALYSIS

Every experiment had three independent repetitions and was carried out in biological triplicates. GraphPad Prism (version 9.0, GraphPad Software Inc., San Diego, CA, USA) was utilised to statistically compare the treated and untreated control groups using one-way analysis of variance (ANOVA) and Tukey's post-hoc test. A p-value of less than 0.05 was considered statistically significant.

4. RESULTS

4.1 Growth features of *A. baumannii:* On LB agar, MacConkey agar, and blood agar plates, distinctive growth patterns of *A. baumannii* were noted. The colonies had morphological characteristics typical of *A. baumannii*. To guarantee the integrity and viability of the isolate, cultures were frequently subcultured and biochemical confirmation was performed before every experimental assay. Furthermore, the effectiveness of 3-FC against *A. baumannii* was evaluated.

4.2 Analysis of antibiotic susceptibility testing: The Clinical and Laboratory Standards Institute (CLSI) recommendations (2022) were followed for conducting antibiotic susceptibility testing. The findings, which are displayed in **Table 1**, show the clinical isolate of *A. baumannii* sensitivity and resistance patterns to the examined drugs.

4.3 Antimicrobial susceptibility analysis: The antimicrobial susceptibility test revealed a clear zone of inhibition measuring 21 mm for the *A. baumannii* clinical isolate (**Figure 1**). These findings suggest that 3-FC possesses antimicrobial potential against *A. baumannii.*

4.4 Inhibitory effect of 3-FC on *A. baumannii:* 3-FC effectively halted the development of *A. baumannii* at the lowest tested concentration of 0.312 mg/mL. Using a two-fold serial dilution experiment (range from 10 mg/mL to 0.019 mg/mL), the antibacterial activity of 3-FC was evaluated; growth inhibition was observed at 0.312 mg/mL. **(Table 2** and **Figure 2A)**.

Additionally, the Alamar Blue assay showed that treatment with 3-FC (0.156 mg/mL) did not significantly affect the metabolic activity of *A. baumannii* compared to the untreated control. Similar fluorescence intensity was observed in both treated and control groups (**Figure 2B**). These results indicate that 3-FC at sub-MIC levels does not alter the metabolic activity of *A. baumannii*.

4.5 Molecular interactions of 3-FC with *A. baumannii* target proteins: The binding potential of 3-FC against key regulatory and biofilm-associated proteins of *A. baumannii* was assessed through molecular docking simulations. Two crystallographic structures were selected as targets: 5HM6, representing the N-terminal domain of *BfmR*, and 4G4Y, corresponding to the *OmpA* peptidoglycan-binding domain. For *OmpA* and *BfmR*, docking study showed binding affinities of −4.4 kcal/mol and −5.8 kcal/mol, respectively (**Table 3A-3B**).

In the 4G4Y complex, 3-FC established π–anion interactions with GLU A:248 via two interaction bonds, while MET A:228 and LYS A:251 contributed π–alkyl interactions, collectively stabilizing the ligand within the binding pocket (**Figure 3A-3C**). In the 5HM6 complex, 3-FC formed a carbon–hydrogen bond with VAL A:109, two π–donor hydrogen bonds with THR A:23, and three π–alkyl interactions involving LEU A:19, PRO A:111, and LYS A:107 (**Figure 4A-4C**).

Taken together, these findings suggest that 3-FC may have promising antibiofilm potential through its favourable interactions with critical *A. baumannii* biofilm-associated proteins. Nevertheless, further experimental validation is warranted to fully elucidate its mechanism of action and therapeutic applicability.

5. DISCUSSION

A. baumannii has emerged as one of the most formidable nosocomial pathogens of the 21st century, owing to its exceptional capacity to acquire MDR and form tenacious biofilms on clinical surfaces. The WHO has consistently classified carbapenem-resistant *A. baumannii* (CRAB) as a critical-priority pathogen a designation reaffirmed in its 2024 priority pathogen list, underscoring the urgent need for novel therapeutic strategies (23,24). In the present study, we evaluated the antibacterial potential of 3-FC against a clinical isolate of *A. baumannii*, integrating antibiotic susceptibility profiling, MIC determination, metabolic activity assessment, and *in-silico* molecular docking analysis.

The antibiotic susceptibility profile of the clinical isolate revealed a broad pattern of resistance, with documented resistance to piperacillin/tazobactam, meropenem, imipenem, ceftriaxone, cefotaxime, and cefixime, and retained sensitivity only to tetracycline. This MDR phenotype is consistent with patterns widely reported in the literature (25). According to Wu et al. (2023), carbapenem resistance rates are close to 90% in Southern Europe, the Middle East, and North Africa, and about 45% of *A. baumannii* isolates worldwide have MDR, with prevalence exceeding 70% in Latin America and the Middle East (26). Similarly, Thacharodi et al. (2024) described CRAB as one of the world's most critically resistant organisms, whose therapeutic options are increasingly limited due to the pathogen's genomic plasticity and resistance island acquisition (27). The resistance landscape in our study reinforces the rationale for exploring non-conventional antibacterial agents such as chromone derivatives.

ZOI of 21 mm was produced by 3-FC in the agar well diffusion assay, against the *A. baumannii* clinical isolate, and the MIC was determined to be 0.312 mg/mL. These findings are broadly aligned with the antibacterial activity previously demonstrated for formylchromone derivatives against Gram-negative pathogens. According to Sathiyamoorthi et al. (2023), 6-bromo-3-formylchromone (6B3FC) and 6-chloro-3-formylchromone (6C3FC) demonstrated MICs of 20 µg/mL against *Vibrio parahaemolyticus* and *V. harveyi*, and dose-consistently decreased significant virulence traits and stopped the formation of biofilms that involved the synthesis of indole, protease activity, and swimming motility (15). Similarly, Boya et al. (2024) showed that three formylchromone derivatives, namely 6-bromo 3-formylchromone, 6-chloro 3-formylchromone, and 3-formyl 6-isopropylchromone, suppressed hemolysis, motility, curli formation, and siderophore production in addition to inhibiting biofilm formation in uropathogenic *Escherichia coli* (UPEC) by 72–96% at 20 µg/mL (28). Although 3-FC in the present study lacks the halogen substituents at the C-6 position that appear to enhance potency in these analogs, its conjugated chromone scaffold retains inherent electron-withdrawing properties that may contribute to membrane disruption and protein interaction. Further structure-activity relationship (SAR) studies exploring halogenated 3-FC derivatives against *A. baumannii* would be an important next step.

The Alamar Blue assay conducted at sub-MIC concentration (0.156 mg/mL) revealed no significant reduction in the metabolic activity of *A. baumannii*, as indicated by comparable fluorescence values between treated and untreated groups. This suggests that 3-FC does not exert a bactericidal effect on cellular metabolic activity at sub-inhibitory levels. These findings are consistent with the MIC results, confirming that antibacterial activity is concentration-dependent (15). The metabolic neutrality observed here may thus position suggest that 3-FC may act as a potential antibiofilm adjunct rather than a conventional bactericidal agent, warranting dedicated biofilm quantification and virulence gene expression studies in future work.

Molecular docking simulations provided mechanistic insight into the potential antibiofilm activity of 3-FC. The two protein targets selected *OmpA* (PDB: 4G4Y) and *BfmR* (PDB: 5HM6) are well-established virulence determinants in *A. baumannii*. *OmpA* is a multifunctional outer membrane protein implicated in biofilm formation, host cell adhesion, immune evasion, and antibiotic resistance, making it a high-value therapeutic target. *BfmR* is the response regulator of the *BfmS/BfmR* two-component system, which governs biofilm development, exopolysaccharide synthesis, and stress adaptation. In the present study, 3-FC demonstrated binding affinities of −4.4 kcal/mol and −5.8 kcal/mol for *OmpA* and *BfmR*, respectively. This affinity observed for *BfmR* is notable, given the central role this regulator plays in coordinating *A. baumannii* biofilm lifecycle.

Overall, despite the encouraging preliminary findings, several limitations of the present study must be acknowledged. First, this investigation was conducted on a single clinical isolate, and the observed antibacterial and docking results may not be fully generalizable across the heterogeneous population of *A. baumannii* clinical strains. Second, although molecular docking simulations provide useful mechanistic insight, they do not account for dynamic conformational changes, solvent effects, or entropic contributions encountered under physiological conditions. Molecular dynamics (MD) simulations and free energy perturbation analyses would be required to validate the stability of the predicted 3-FC *OmpA* and 3-FC *BfmR* complexes. Third, dedicated antibiofilm assays, including crystal violet quantification and confocal microscopy of biofilm architecture, were not performed in the current study, and such experiments are essential to confirm the *in-silico* predictions.

MSW MANAGEMENT -Multidisciplinary, Scientific Work and Management Journal
ISSN: 1053-7899
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6. CONCLUSION

The present study demonstrates that 3-FC exhibits notable antibacterial activity against a MDR clinical isolate of *A. baumannii*, with a MIC of 0.312 mg/mL and a zone of inhibition of 21 mm. Molecular docking analysis revealed favorable binding affinities of 3-FC toward *A. baumannii* target proteins, including *OmpA* (−4.4 kcal/mol) and *BfmR* (−5.8 kcal/mol). These interactions suggest a potential association with proteins implicated in bacterial physiology and regulatory pathways. However, the functional implications of these interactions remain to be experimentally validated. Therefore, further studies, including targeted phenotypic assays and molecular dynamics simulations, are required to elucidate the precise mechanism of action and therapeutic relevance of 3-FC.

ACKNOWLEDGMENTS

Nil

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest in relation to the submitted work.

AUTHORS' CONTRIBUTION

Each of the authors mentioned has made significant contributions to the research

ETHICS STATEMENT

Not applicable

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ISSN: 1053-7899
Vol. 36 Issue 1, 2026, Pages 6258-6262

Tables

Table 1. Antibiotic susceptibility of clinical isolate of *A. baumannii.*

Antibiotic Name and Disk Potency (µg)	Zone of Inhibition (Clinical Isolate)	Clinical Isolate Interpretation (CLSI)
Piperacillin/Tazobactam (100/10)	12 mm	Resistant
Tetracycline (30)	25 mm	Sensitive
Cefixime (5)	No zone	Resistant
Meropenem (10)	No zone	Resistant
Imipenem (10)	No zone	Resistant
Ceftriaxone (30)	No zone	Resistant
Cefotaxime (30)	13 mm	Resistant

Table 2. MIC of 3-FC against *A. baumannii*.

Concentration of 3-FC (mg/mL)	*A. baumannii* (Clinical isolate)
10	−
5	−
2.5	−
1.25	−
0.625	−
0.312	−
0.156	+
0.078	+
0.039	+
0.019	+
Control	+

(−) No visible growth; (+) Visible bacterial growth.

Table 3. Molecular docking results of 3-FC against *A. baumannii* target proteins. Binding affinities (kcal/mol) and root mean square deviation values (rmsd/ub and rmsd/lb) for the top docking poses of 3-FC against *OmpA* (4G4Y) and *BfmR* N-terminal domain (5HM6). The best-ranked pose for each target is highlighted (binding affinity: −4.4 kcal/mol and −5.8 kcal/mol, respectively).

Ligand	Binding Affinity	rmsd/ub	rmsd/lb
4G4Y_A_87112_uff_E=112.49	-4.4	0	0
4G4Y_A_87112_uff_E=112.49	-4.2	4.786	1.644
4G4Y_A_87112_uff_E=112.49	-4.1	4.356	1.59
4G4Y_A_87112_uff_E=112.49	-4	4.295	1.982
4G4Y_A_87112_uff_E=112.49	-4	4.677	1.835
4G4Y_A_87112_uff_E=112.49	-3.9	16.593	14.716
4G4Y_A_87112_uff_E=112.49	-3.8	5.185	2.93
4G4Y_A_87112_uff_E=112.49	-3.8	17.07	15.654
4G4Y_A_87112_uff_E=112.49	-3.7	5.116	2.27
5HM6_A_87112_uff_E=112.49	-5.8	0	0
5HM6_A_87112_uff_E=112.49	-5.4	4.745	1.731
5HM6_A_87112_uff_E=112.49	-5.4	2.578	1.22
5HM6_A_87112_uff_E=112.49	-5.4	1.06	1.045
5HM6_A_87112_uff_E=112.49	-5.3	4.307	1.674
5HM6_A_87112_uff_E=112.49	-5.2	4.957	1.59
5HM6_A_87112_uff_E=112.49	-5.1	3.064	1.749
5HM6_A_87112_uff_E=112.49	-5.1	5.029	2.798
5HM6_A_87112_uff_E=112.49	-4.9	3.479	1.773

Figures

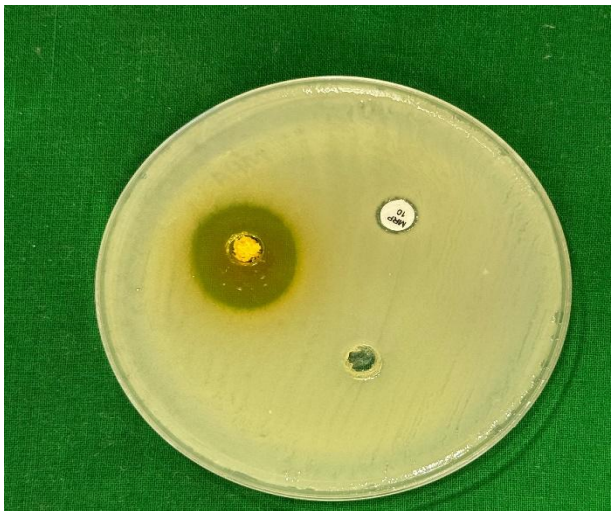

Figure 1. Antimicrobial activity of *A. baumannii* showing zones of 21 mm in response to 3-FC treatment.

MSW MANAGEMENT -Multidisciplinary, Scientific Work and Management Journal
ISSN: 1053-7899
Vol. 36 Issue 1, 2026, Pages 6258-6262

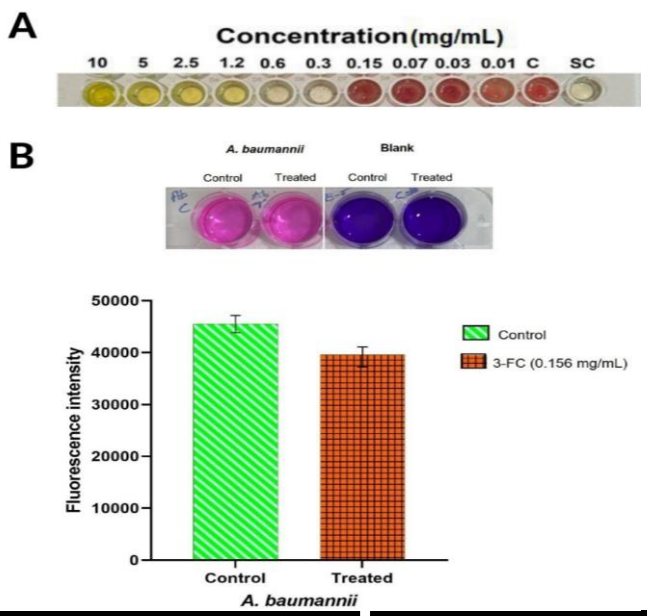

Figure 2. Antibacterial activity of 3-FC against *A. baumannii*. (A) Two-fold serial dilution assay showing the minimum inhibitory concentration (MIC) of 3-FC (10-0.019 mg/mL) against *A. baumannii* **(B)** Alamar Blue assay comparing the metabolic activity of untreated (control) and 3-FC-treated (0.156 mg/mL -sub-MIC) *A. baumannii*. Representative well images (top) and fluorescence intensity values (bottom) are shown. Data are expressed as mean ± SD.

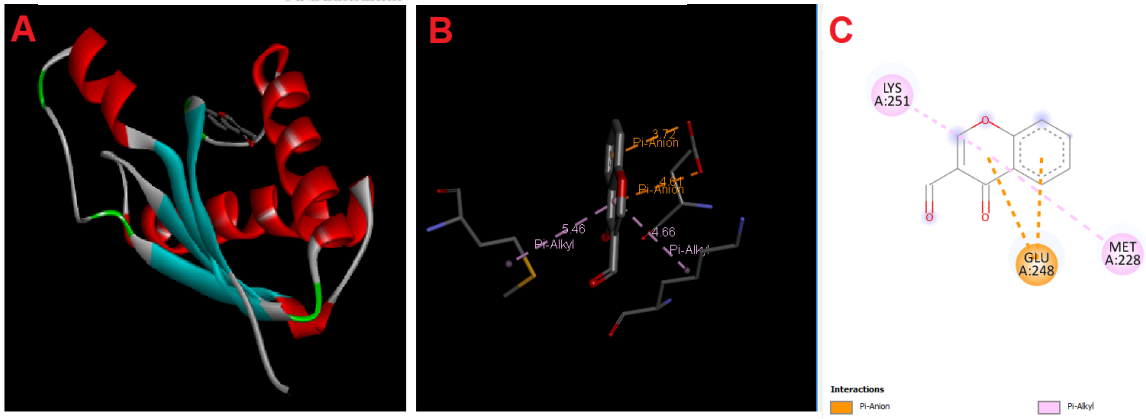

Figure 3. Molecular docking interactions of 3-FC within the binding pocket of 4G4Y. (A) Overall docked 3D view **(B)** 3D close-up of key binding interactions. **(C)** 2D interaction map showing π–anion interactions with GLU A:248 and π–alkyl interactions with MET A:228 and LYS A:251.

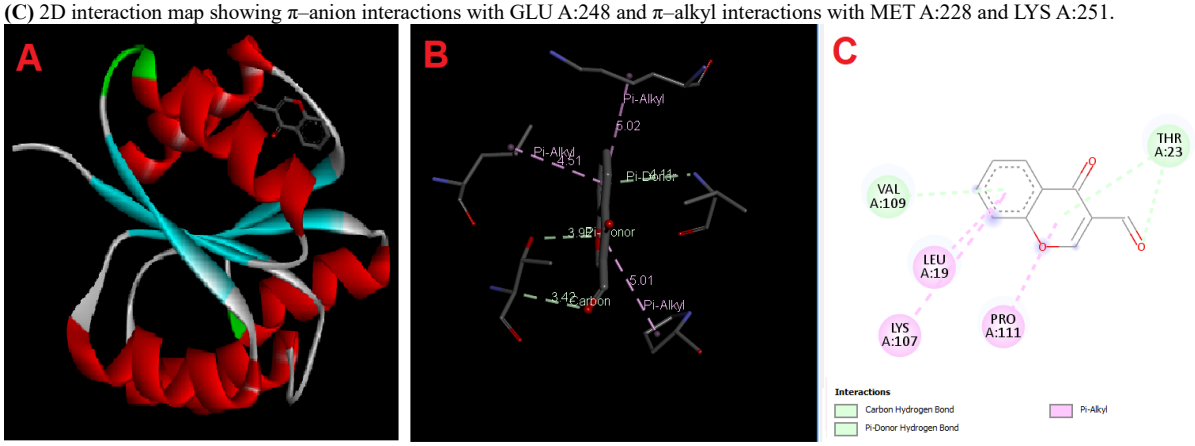

Figure 4. Molecular docking interactions of 3-FC within the binding pocket of 5HM6. (A) Overall docked 3D view **(B)** 3D close-up of key binding interactions. **(C)** 2D interaction map showing a carbon–hydrogen bond with VAL A:109, π–donor hydrogen bonds with THR A:23, and π–alkyl interactions with LEU A:19, PRO A:111, and LYS A:107.