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Mechanistic Insights into Antibiofilm Activity Against Meropenem-Resistant *Acinetobacter baumannii*

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Abstract

***Acinetobacter baumannii* is considered the third most common** cause of catheter-associated urinary tract infections (UTIs). Its ability to form biofilms and produce urease contributes to catheter blockage, enhancing its virulence and reducing the effectiveness of antibiotics. UTIs caused by *A. baumannii* are increasingly concerning, and meropenem remains a last-resort treatment for infections caused by this pathogen. However, the emergence of meropenem-resistant (MER-R) *A. baumannii* has severely compromised its clinical effectiveness. This study aimed to investigate the antibiofilm activity and underlying mechanisms associated with MER-R *A. baumannii*. Urine samples were collected from 102 patients referred to Baghdad Medical City and immediately placed in sterile tubes before initial culture on MacConkey agar. Identification and confirmation of bacterial isolates were performed using the Vitek® 2 Compact system with a Gram-negative (GN) identification card, which includes 48 biochemical tests. Antibiotic susceptibility testing and biofilm activity assays of *A. baumannii* were also conducted. The results confirmed the isolation of *A. baumannii*. Antibiotic susceptibility testing revealed that a sub-minimum inhibitory concentration (sub-MIC) of 62 µg/mL meropenem exhibited a significant biofilm-inhibitory effect against multidrug-resistant wild-type *A. baumannii* strains. Strong biofilm-producing isolates were incubated with 1 mL of sub-MIC colistin for 24 and 48 hours at 37 °C. Meropenem was further evaluated for its ability to inhibit biofilm formation by *A. baumannii*. Conclusion: This study highlighted the mechanisms underlying antibiofilm activity against meropenem-resistant *A. baumannii*. Targeting biofilm formation and associated virulence factors can significantly reduce bacterial colonization and catheter obstruction, offering a promising strategy to enhance treatment efficacy and improve patient outcomes. These findings provide valuable insights for developing alternative therapeutic approaches against multidrug-resistant *A. baumannii* infections.

Keywords: *A. baumannii*, Biofilm, Meropenem, Antibiofilm.

Introduction

A common Gram-negative, non-flagellated coccobacillus bacteria that is frequently isolated from the environment is *Acinetobacter baumannii*. Infections acquired in hospitals and the community are caused by this opportunistic pathogen in human medicine. (1) The study of *A. baumannii* as a whole is made more difficult by the substantial genetic variation among isolates, which is caused by a flexible genome. As a

result, *A. baumannii* poses a contemporary, global threat that the public health community must continually monitor (2). The study of *A. baumannii* as a whole is made more difficult by the considerable genetic variation among isolates, which is caused by a flexible genome (3). More recently, military troops wounded in the wars in Iraq and Afghanistan have contracted a variety of infectious diseases from *A. baumannii*.

Some of the most notable advances in the understanding of this fascinating organism over the past decade include the current taxonomy and species identification, challenges in susceptibility testing, mechanisms of antibiotic resistance, global epidemiology, the clinical impact of infection, the connection between host-pathogen relationships, infection control, and treatment considerations (4). By compiling the most recent data on bacterial virulence factors, their cellular targets, and genetic regulation, covered a wide range of factors critical to this pathogen's success, such as membrane proteins and cell surface adaptations that support immune evasion, mechanisms for absorption of nutrients, and community interactions (5). In order to determine the most promising treatment plan, current efforts concentrate on targeting every antibiotic resistance mechanism identified in *A. baumannii*. Although therapeutic strategies based on bacteriophages and artilysin have been shown to be successful, more investigation into their clinical application is necessary (6). The intrinsic antimicrobial resistance mechanisms of ABC organisms could either be expressed constitutively or in accordance with antibiotic pressure. The percentage of resistance to all antimicrobial agents in four major classes of antimicrobials increased between 1995 and 2004, according to an analysis of susceptibility data for ABC organisms (7). Li-Kuang and companions studied *A. baumannii* clinical isolates. According to Previous research, 73.6% of these isolates are resistant to quinolones (ciprofloxacin and levofloxacin), 71.3% to sulfonamides, and more than half (50–70%) to cephalosporins (cefazidime and cefepime), b-lactam/beta-lactamase inhibitor combinations (tazobactam–piperacillin), and carbapenems (doripenem, imipenem, and meropenem). Remarkably, only 26.7% of patients had resistance to the tigecycline treatment. Additionally, according to another previous study, there was no evidence of colistin resistance (8).

A protein associated with biofilm (BAP) is found in the outer membrane of *A. baumannii* and is necessary for biofilm maturation. It increases the biofilm's thickness and volume, promoting intercellular adhesion. A locus in *A. baumannii* codes for proteins that produce cell-associated poly-β-(1-6)-N-acetylglucosamine (PNAG) polysaccharide. This is essential for maintaining stability under stressful conditions (9).

Carbapenem-resistant *A. baumannii* (CRAB) isolates have become more common, leading to longer hospital admissions and higher mortality rates. Carbapenems are a prevalent treatment for *A. baumannii* infections (10). Imipenem acts against Gram-negative bacteria by crossing the outer cell envelope and binding to certain penicillin-binding proteins (PBPs) with high affinity. This is especially true for meropenem and imipenem. Imipenem's broad-spectrum impact is also due to resistance to various β-lactamases (11).

The objectives of this study were to evaluate the antibiofilm activity of meropenem against meropenem-resistant *Acinetobacter baumannii* and to examine the underlying mechanisms of biofilm inhibition at sub-minimum inhibitory concentrations (sub-MICs).

Materials and Methods

Ethical approval

This study was conducted in accordance with ethical standards and was approved by the Ethics Committee of the College of Medicine, Al-Iraqia University (Approval No. FM.SA/97, dated 28 April 2026

Sample collection

A total of 102 clinical specimens were collected from patients referred to Baghdad Medical City, Baghdad, between October and December 2025. Urine samples were obtained using sterile cotton swabs and immediately placed into sterile tubes containing transport medium before being transferred to the laboratory for microbiological analysis.

Isolation and Identification

Acinetobacter baumannii was initially identified based on its growth characteristics on MacConkey agar (Lab, England). Clinical specimens were cultured and aerobically incubated at 37 °C for 24 hours. Pale, non-lactose-fermenting colonies were selected and subcultured for further examination. Preliminary identification was performed using oxidase, catalase, and Gram staining assays. Bacterial isolates were subsequently confirmed using the Vitek® 2 Compact system with a Gram-negative (GN) identification card, which includes 48 biochemical tests (12).

Antibiotic susceptibility by Kirby-Bauer disk diffusion test

Briefly, Mueller-Hinton agar plates were used to cultivate verified *A. baumannii* isolates, and the consistency was adjusted to a 0.5 McFarland standard. The bacterial suspension was uniformly inoculated onto the agar surface using sterile cotton swabs. Antibiotic discs include several classes, such as β-lactams (ticarcillin, piperacillin/tazobactam, ceftazidime, cefepime), mono antibiotics (aztreonam), carbapenems (imipenem, meropenem), and aminoglycosides (amikacin, gentamicin, tobramycin). The inoculated plates received treatments with polymyxin (colistin) and fluoroquinolone (ciprofloxacin). For eighteen to twenty-four hours, plates were aerobically incubated at 37 °C. Based on CLSI assessment criteria, isolates were assigned as sensitive, moderate, or resistant, and zones of inhibition were established in millimeters. For every antibiotic tested, a percentage of resistant and sensitive isolates was computed to express the results (13).

Biofilm activity of *Acinetobacter baumannii*

Sterile brain heart infusion (BHI) broth was used to adjust 18-hour-old cultures of the selected strains to a final concentration of 10^1 CFU/mL. For biofilm formation, 20 µL of the bacterial suspension, 180 µL of BHI, and 1% glucose were added to each well of sterile 96-well polystyrene microtiter plates. Wells containing only BHI served as negative controls. Plates were incubated at 37 °C for 24 hours to allow biofilm development. Biofilm biomass was quantified using the crystal violet assay. After incubation, wells were air-dried, fixed with 150 µL methanol for 15 minutes, and gently washed with 1× PBS. Biofilms were then stained with 0.2% (w/v) crystal violet for 15

minutes, rinsed with distilled water, and air-dried. The bound dye was dissolved in 95% ethanol, and biofilm biomass was measured by absorbance at 630 nm (13).

Minimum inhibitory concentration of Meropenem

The cationic adjusted Mueller–Hinton broth (CAMHB) microdilution method was used to calculate the minimum inhibitory concentration (MIC) of colistin. For colistin manufactured on CAMHB 96-well microtiter plates, Colistin Eas, procured from Zhejiang, China's Ltd., was diluted twice and put through a series of two-fold dilutions that varied from 1000 to 62.5 µg/mL. Each well was filled with a final bacterial suspension of 1.5 × 106 CFU/mL, and the plate was then treated with colistin for 18 hours at 37°C. The antimicrobial susceptibility test was interpreted using the 2020 CLSI breakpoint for antibiotics (intermediate < 2 µg/mL; resistant > 4 µg/mL), and each MIC test was independently verified as a duplicate (14).

Screening for antibiofilm activity of Meropenem

Culture preparation: Bacterial cultures were grown in brain-heart infusion (BHI) broth for 24 hours at 37 °C. Their concentration was then adjusted to 1.5×10^5 CFU/mL using a Densi CHEK device. Plate setup: Each well of a 96-well polystyrene microplate was filled with 180 µL of BHI containing 1% glucose. Then, 10 µL of the standardized bacterial culture was added to each well. For testing colistin, 200 µL of BHI containing bacteria and colistin at its MIC was added to the wells. Wells with only sterile medium were used as negative controls. Removing planktonic cells: During incubation, free-floating (planktonic) bacteria were gently removed by washing the wells four times with 0.2 mL of phosphate-buffered saline (PBS, pH 7.2).Biofilm staining and measurement: The remaining attached biofilms were fixed with 95% methanol and stained with 0.2% crystal violet. Excess stain was washed away with distilled water. After drying, the bound dye was dissolved in 200 µL of 95% ethanol, and the amount of biofilm was measured by optical density (OD) at 630 nm using a microplate reader. Calculating biofilm inhibition: The effect of colistin on biofilm formation was expressed as % biofilm inhibition, calculated with the formula: % biofilm inhibition = 100- (OD of Colistin treated cells/ OD sample) *100

Results

A 33 isolates (32.35%) of the 102 bacterial specimens taken from patients samples were found to be *Acinetobacter baumannii*. *A. baumannii* developed circular, mucoid, non-lactose-fermenting colonies of Gram-negative bacilli on MacConkey agar (Figure. 1). The isolates were oxidase-negative and catalase-positive. The Vitek® 2 identification procedure was used to confirm that all 33 isolates were *A. baumannii*.

Identification by the VITEK2-System

The antibiotic susceptibility of all 33 *Acinetobacter baumannii* isolates against Conventional biochemical tests were used to further characterize all isolates that developed on the selective media, and it was discovered that they were oxidase negative

and catalase positive. Furthermore, every isolate showed that *A. baumannii* can thrive at 42 °C. The identification probabilities ranging from 98% to 99%, the Vitek® 2 system were used to confirm the 33 isolates. Nine antimicrobial agents from four distinct antibiotic classes showed (Figure. 2). The Clinical and Laboratory Standards Institute (CLSI, 2022) recommendations were followed when interpreting the susceptibility results (Table 2).

Antibiotic Susceptibility Profile of *Acinetobacter baumannii*

Acinetobacter baumannii isolates had a high degree of multidrug resistance, according to antibiotic susceptibility testing. Significant resistance was found to β-lactam antibiotics, such as ticarcillin and piperacillin (66% resistance each), although moderate resistance was found to ceftazidime and piperacillin/tazobactam (around 37%). Cefepime and aztreonam had similar resistance rates (36%), suggesting that monobactams and extended-spectrum cephalosporins are less effective. The activity of carbapenems varied; imipenem showed equivalent levels of sensitivity and resistance (63%), whereas meropenem showed more resistance (83%). The efficiency of aminoglycosides varied; gentamicin showed considerable resistance (90%) while amikacin showed comparatively good sensitivity (72%). Only 10% of isolates showed ciprofloxacin resistance, indicating a lower level of fluoroquinolone resistance. With 89% of isolates demonstrating sensitivity, colistin continued to be the most effective antibiotic, underscoring its significance as a last-resort therapy option against multidrug-resistant *A. baumannii* (Figure.3).

Table 1: Isolation percentage of bacteria according to specimen source

Specimen source	Positive n. (%) from 33 Isolates	Positive n. (%) from 102 Specimens
Urine	33 (100%)	(32.35%)

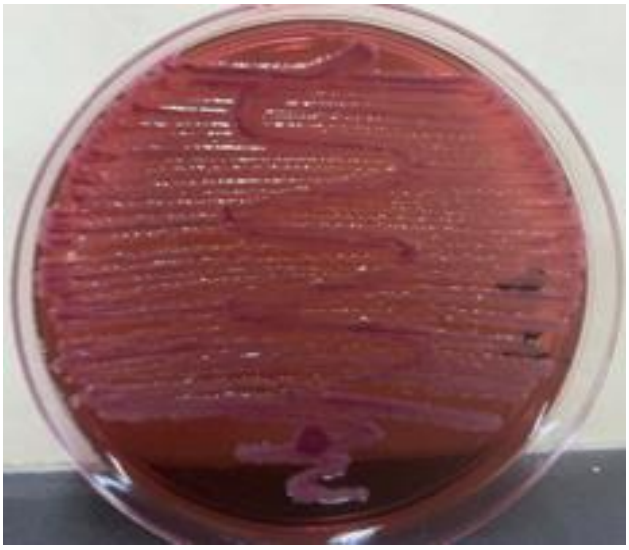
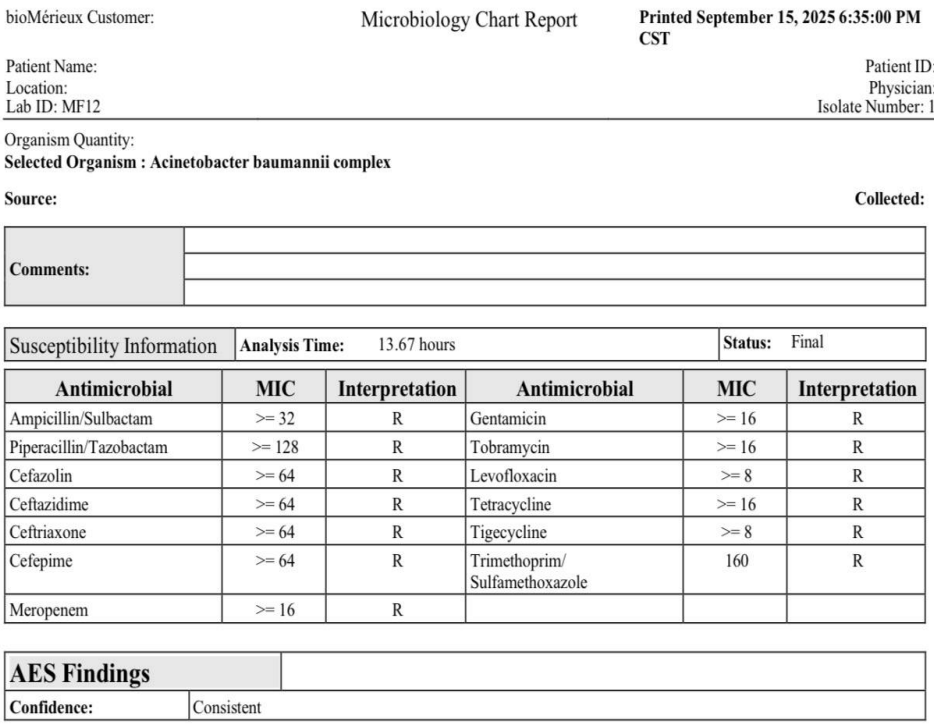


Figure.1: Shows *Acinetobacter baumannii* colonies on MacConkey agar

Table. 2: Antibiotic Susceptibility by Vitek® 2 system

Antimicrobial	MIC (µg/mL)	Interpretation
Ampicillin/Sulbactam	>=32	R
Piperacillin/Tazobactam	>=128	R
Cefazolin	>=64	R
Ceftazidime	>=64	R
Ceftriaxone	>=64	R
Cefepime	>=64	R
Meropenem	>=16	R
Gentamicin	>=16	R
Tobramycin	>=16	R
Levofloxacin	>=8	R
Tetracycline	>=16	R
Tigecycline	>=8	R
Trimethoprim/Sulfamethoxazole	160	R



bioMérieux Customer: Microbiology Chart Report **Printed September 15, 2025 6:35:00 PM CST**

Patient Name: Patient ID:
Location: Physician:
Lab ID: MF12 Isolate Number: 1

Organism Quantity:
Selected Organism : Acinetobacter baumannii complex

Source: **Collected:**

Comments:	

Susceptibility Information	**Analysis Time:** 13.67 hours	**Status:** Final

Antimicrobial	MIC	Interpretation	Antimicrobial	MIC	Interpretation
Ampicillin/Sulbactam	>= 32	R	Gentamicin	>= 16	R
Piperacillin/Tazobactam	>= 128	R	Tobramycin	>= 16	R
Cefazolin	>= 64	R	Levofloxacin	>= 8	R
Ceftazidime	>= 64	R	Tetracycline	>= 16	R
Ceftriaxone	>= 64	R	Tigecycline	>= 8	R
Cefepime	>= 64	R	Trimethoprim/ Sulfamethoxazole	160	R
Meropenem	>= 16	R			

AES Findings	
Confidence:	Consistent

Figure. 2: Identification of *Acinetobacter baumannii by* VITEK 2 - System

Antibiotic Susceptibility Profile of *Acinetobacter baumannii*

Acinetobacter baumannii isolates had a high degree of multidrug resistance, according to antibiotic susceptibility testing. Significant resistance was found to β-lactam antibiotics, such as ticarcillin and piperacillin (66% resistance each), although moderate resistance was found to ceftazidime and piperacillin/tazobactam (around 37%). Cefepime

and aztreonam had similar resistance rates (36%), suggesting that monobactams and extended-spectrum cephalosporins are less effective. The activity of carbapenems varied; imipenem showed equivalent levels of sensitivity and resistance (63%), whereas meropenem showed more resistance (83%). The efficiency of aminoglycosides varied; gentamicin showed considerable resistance (90%) while amikacin showed comparatively good sensitivity (72%). Only 10% of isolates showed ciprofloxacin resistance, indicating a lower level of fluoroquinolone resistance. With 89% of isolates demonstrating sensitivity, colistin continued to be the most effective antibiotic, underscoring its significance as a last-resort therapy option against multidrug-resistant *A. baumannii* (Figure.3).

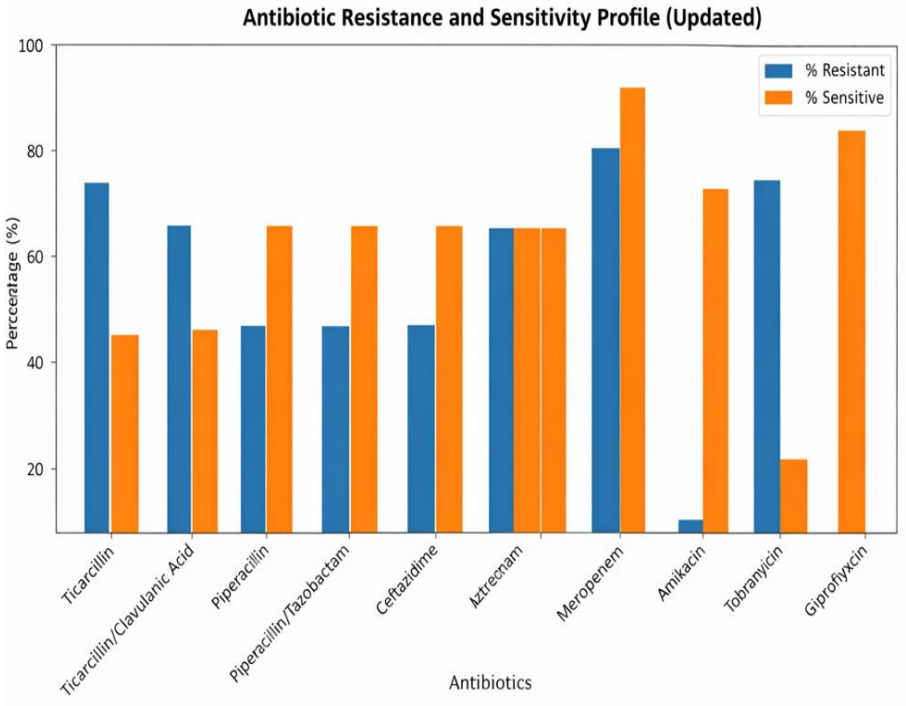


Figure. 3: Percentage of Antibiotic profile for *Acinetobacter baumannii*

Determination of biofilm formation before Meropenem treatment

A quantitative technique utilizing microtiter plates (MTP) was used to evaluate the MDR *A. baumannii* isolates' capacity to generate biofilms (n = 33). The results showed that isolates of *A. baumannii* biofilm production are strong, 13 % and 5 % generate weak biofilm, 10% produce moderate biofilm (Figures 4 and 5).

Minimum inhibitory concentration of Meropenem

The lowest MIC of Meropenem for ten *A. baumannii* isolates (A-F) was determined for each isolate (Table. 3 and Figure. 6).

Figure. 4: Biofilm formation by *Acinetobacter baumannii* isolates was assessed using the microtiter plate method.

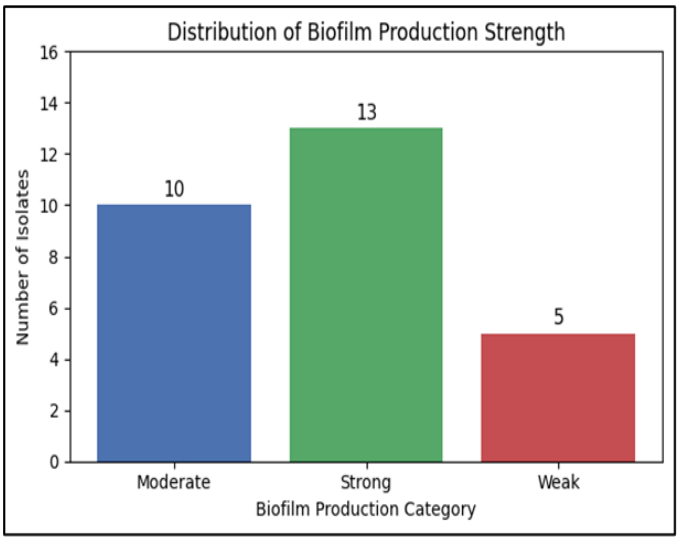


Figure. 5: *Acinetobacter baumannii* isolates' biofilm values as a percentage using the microtiter plate method

Table.3: The minimal inhibitory concentration of Meropenem

Bacterial isolate NO.	MIC of Meropenem (µg/ml)
A	125
B	250
C	62
D	250
F	62

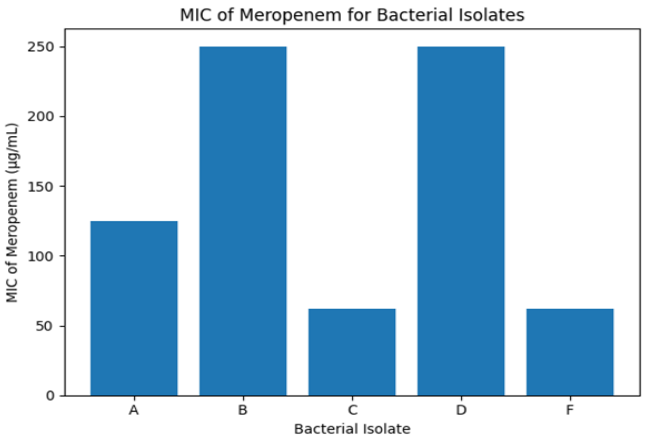


Figure.6: Zone of Bacterial Inhibition in mm Treated with Different Concentrations (in µg/mL) of Meropenem against ten *A. baumannii*.

Determination of biofilm formation after Meropenem treatment

Measurement of optical density (OD) for the same *Acinetobacter baumannii* strains demonstrated a marked reduction in biofilm production, shifting from strong to weak biofilm formation after 24 h of incubation at 37 °C in the presence of meropenem at its minimum inhibitory concentration (MIC). The percentage inhibition of biofilm formation caused by meropenem was calculated using the following formula: Biofilm inhibition (%)=100−(OD before treatment OD after treatment×100)
Moreover, the results showed that meropenem exhibited a significant inhibitory effect on biofilm formation in the tested *A. baumannii* isolates (Figure 7).

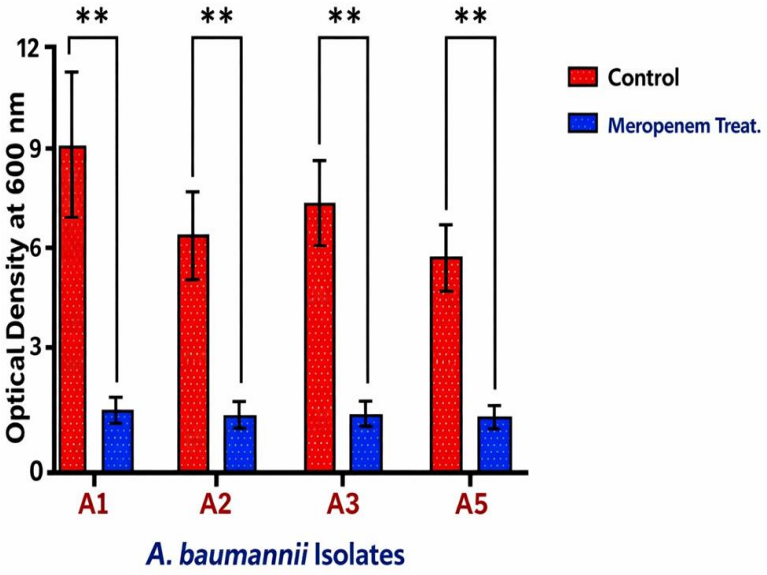


Figure. 7: Antibiofilm activity of Meropenem

Discussion

Exposure to sub-minimum inhibitory concentrations (sub-MICs) of meropenem significantly reduced biofilm biomass in the tested *A. baumannii* strains. This results indicated that meropenem, even at concentrations below the MIC, can disrupt biofilm formation, potentially by interfering with bacterial cell communication or the production of extracellular polymeric substances (EPS). Similar antibiofilm effects of sub-MICs have been reported in *A. baumannii* and other Gram-negative pathogens. Previous studies suggest that downregulation of the biofilm-associated protein (BAP) or disruption of quorum-sensing pathways may underlie the observed reduction in biofilm formation at sub-MICs (15). Understanding these mechanisms is critical for developing strategies to combat biofilm-associated infections, particularly in multidrug-resistant bacteria where conventional antibiotics are often ineffective. The findings highlight the challenges of treating catheter-associated UTIs caused by meropenem-resistant *A. baumannii* (16). Importantly, the antibiofilm activity of meropenem at sub-MICs supports the potential use of adjuvants or combination therapies to enhance antibiotic efficacy. This approach aligns with current recommendations for biofilm-targeted strategies in the management of multidrug-resistant infections (17). Antibiotics have been essential in treating severe infections and have saved countless lives. However, extensive evidence indicates that the overuse, misuse, and inappropriate prescribing of antibacterial agents have driven the emergence of antibiotic-resistant bacterial strains, which are associated with serious adverse clinical outcomes (18). Elevated mortality rates in intensive care units associated with *A. baumannii* bacteremia are often due to severe comorbidities and potential co-infections, whereas community-acquired *A. baumannii* infections generally result in lower fatality rates. Healthcare-associated environments are strongly linked with *A. baumannii* infections (19). A retrospective study from Thailand reported an increase in carbapenem-resistant *A. baumannii* bloodstream infections (BSIs) during the COVID-19 pandemic. Furthermore, no direct correlation was observed between hospital-wide carbapenem use and resistance, suggesting that strict infection control measures may play a more critical role in limiting *A. baumannii* spread than antibiotic stewardship alone (20). First-line treatments for *A. baumannii* infections include meropenem, imipenem, gentamicin, trimethoprim/sulfamethoxazole, levofloxacin, colistin, tobramycin, amikacin, and imipenem/relebactam, while second-line therapies include tigecycline and ampicillin-sulbactam. A similar approach is used for urinary tract infections, as A. baumannii is frequently isolated from urine cultures in hospital (21). One of the most significant virulence traits of *A. baumannii* is its ability to form biofilms, which enhances bacterial survival in healthcare settings and is strongly associated with increased antibiotic resistance (22). Biofilm formation complicates treatment, promotes person-to-person transmission, and contributes to hospital outbreaks. Biofilms can develop on various hospital surfaces and medical equipment, allowing bacteria to persist and survive even under adverse environmental conditions or antimicrobial pressure (23). The correlation between genetic background and phenotypic adaptability in *A. baumannii* is illustrated by strain-specific variability in biofilm formation and motility under antibiotic stress (24). Meropenem has anti-motility and anti-biofilm properties, indicating that it may be used to direct focused infection control tactics against high-risk *A. baumannii* clones (25). Because of its multidrug resistance, *Acinetobacter baumannii* has been

identified as a priority pathogen worldwide and is a major cause of chronic opportunistic infections in clinical settings. Its pathogenicity and antimicrobial resistance are mostly determined by quorum sensing and biofilm development

Conclusions

This study emphasized on the antibiofilm potential of meropenem against meropenem-resistant *Acinetobacter baumannii* and provides insights into the underlying mechanisms of biofilm inhibition at sub-minimum inhibitory concentrations (sub-MICs). Exposure to sub-MICs of meropenem significantly reduced biofilm biomass, likely through interference with bacterial cell communication, extracellular polymeric substance (EPS) production, and downregulation of biofilm-associated proteins. These findings underscore the critical role of biofilm formation in the persistence and virulence of multidrug-resistant *A. baumannii*, particularly in catheter-associated urinary tract infections. Targeting biofilm formation represents a promising strategy to enhance the effectiveness of existing antibiotics, potentially through adjuvant or combination therapies. By disrupting biofilm development and associated virulence factors, it is possible to reduce bacterial colonization, limit the spread of infection, and improve patient outcomes. Overall, this study provides valuable mechanistic insights that can inform the development of novel therapeutic approaches against multidrug-resistant *A. baumannii* infections.

Declarations

Acknowledgment

None

Ethics statement

The author approved that this research follows the journal's Attached Ethic Approval guidelines as appeared on the journal's author guidelines page.

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None

Competing interest’s statement

The authors declare that they have no conflict of interest.

Author contributions

NKI and **SK** provided the concepts, data analysis, and writing of the manuscript; **NKI** , **MEA** worked with data collection and analysis; **NKI** revised the manuscript and analyzed the data.

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