

Study of fluoroquinolone resistance genes in *Acinetobacter baumannii* isolated from clinical sources

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Article history

Received: 25/04/2026

Revised: 13/05/2026

Accepted: 31/05/2026

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Abstract: *Acinetobacter baumannii* is a common multidrug-resistant bacteria that causes hospital-acquired illnesses. This research attempted to identify fluoroquinolone resistance genes in **Methods:** *A. baumannii* isolates recovered from various clinical materials, including burns, blood, sputum, urine, and diabetic foot infections collected during September to December 2025 in Al-Najaf City, involved 199 (53%) male and 176 (47%) female subjects. The bacterial culture results indicated that out of 375 specimens, 157 (41.9%) exhibited single growth, 28 (7.4%) displayed mixed growth, and 190 (50.7%) showed no bacterial growth. **Results :** This study identified 155 instances (41.33%) of gram-negative bacteria. The Vitek-2 system identified 28 isolates (7.46%) as *A. baumannii*. The highest isolation rate occurred in burn patients at 17 (4.53%), followed by 4 (1.07%) isolates from patients with respiratory tract infections, and 3 (0.8%) from blood source records. Diabetic foot infections and urinary tract infections were documented in two instances (0.53%). The antimicrobial susceptibility test results revealed that all 28 (100%) isolates demonstrated resistance to Cefoxitine, Ceftazidime, and Ciprofloxacin. Additionally, 27 (96.42%) of the isolates were resistant to meropenem, while 26 (92.85%) exhibited resistance to tobramycin and Ampicillin-Sulbactam. Concurrently, 89.28% of the isolates were resistant to gentamicin. The PCR analysis results indicated that the *qnrA*, *qnrB*, and *qnrS* genes were identified in 11 (39%), 7 (25%), and 21 (75%) of the *A. baumannii* isolates, respectively. **Conclusion:** The local isolates exhibited multidrug resistance and harbored diverse fluoroquinolone resistance genes, complicating treatment.

Keywords: *Acinetobacter baumannii*, Qnr gene, Antibacterial agent and PCR

1.Introduction

The *A. baumannii* is a gram-negative coccobacillus previously regarded as an opportunistic pathogen, significantly contributing to healthcare-associated illnesses. This bacterium is recognized for its resilience to extreme environmental conditions, including prolonged life on desiccated surfaces and human skin, which enhances its persistence and dissemination in hospitals and other healthcare settings [2,3,4]. *A.*

baumannii induces infections in nosocomial environments, which are varied and may include hospital-acquired pneumonia, ventilator-associated pneumonia, urinary tract infections, meningitis, bacteremia, as well as gastrointestinal and skin/wound infections [5]. *A. baumannii* has inherent resistance to many antimicrobials and possesses a notable capacity to develop further resistance to numerous kinds of antimicrobials, including carbapenems, aminoglycosides, and fluoroquinolones. This has resulted in the evolution

of multidrug-resistant, extensively drug-resistant, and pan-resistant strains of *A. baumannii*. Multidrug resistance, particularly Carbapenem resistance in *A. baumannii*, constitutes a worldwide healthcare issue. The mechanisms of antibiotic resistance may be classified into three categories, the first of which involves lowering membrane permeability to obstruct access to the target. Secondly, bacteria may safeguard the antibiotic target by genetic mutation or post-translational modification; finally, antibiotics may be directly inactivated via hydrolysis or modification [9]. So current work aimed to screening of some fluoroquinolone resistance genes in *A. baumannii* isolated from clinical sources.

2.Methodology

Collection of Specimens

This investigation used a cross-sectional analytic design, including 375 clinical specimens from patients with diverse conditions, with 75 specimens for each source (burns, diabetic foot infections, urine , blood, and sputum).. All specimens were obtained from Al-Sadr Medical City, Burn Center, Al-Najaf Al-Ashraf Teaching Hospital and and Al-Zahraa Teaching Hospital For Maternity and Pediatrics and Private Laboratories in Al-Najaf City, in addition to many private clinical laboratories. Informed consent was obtained from all patients prior to specimen collection.

Isolates and bacterial diagnosis

All clinical specimens were collected and promptly sent to the microbiology laboratory. All specimens were cultivated on selective and differential media, including MacConkey agar, and Blood agar, to promote the development and first differentiation of bacterial pathogens. Subsequently, isolates were subcultured on Chromogenic agar and incubated at 37 °C for 24 hours to enhance colony visibility for presumptive identification of *A. baumannii*. After incubation, the morphology of colonies on agar plates was meticulously examined, recording characteristics relevant to bacterial differentiation. Identification of *A. baumannii* performed using gram stain, culture characteristics, motility citrate, oxidase and catalase tests, and corroborated by the Vitek-2 automated system [10].

Antimicrobial agents susceptibility

All *A. baumannii* isolates were evaluated against a panel of antibiotics disks (Bioanalyse, Turkey) using disk diffusion on sterile Mueller Hinton agar medium. The suspensions of all tested isolates were prepared according to the 0.5 McFarland standard, including Ampicillin-Sulbactam (A/S, 10/10 mg), Cefoxitin (FOX, 30 mg), Ceftazidime (CAZ, 30 mg), Meropenem (MEM, 10 mg), Gentamicin (GEN, 10 mg), Tobramycin (TOB, 10 mg), and Ciprofloxacin (CIP, 5 mg). following the Kirby–Bauer technique [11]. The zone diameters were implemented according to the guidelines of the Clinical and Laboratory Standards Institute [12].

DNA extraction and PCR assay

In accordance with the instructions provided by a manufacturing company, a genomic DNA extraction micro kit (Favorgen, South Korea) was used to extract nucleic acids from 28 clinical isolates of *A. baumannii*. This was carried out in accordance with the manufacturer's protocol. After ensuring the complete integrity of the DNA sample by storing it in a deep freezer at -20 degrees Celsius, a PCR analysis was performed to investigate the genes listed in Table 1. The gel documentation apparatus was used for the migration of PCR amplification bands on 1% agarose, subsequently coloring the bands with Green Safe dye (Sigham, Iran).

Table 1. Primer sequence and thermocycling conditions

Prime r	Na me	Sequence (5' to 3')	Anneal ing (C°)	Produ ct size (bp)	Referen ce
qnrA	F	GATAAAGTTTTTCAGCAAGAGG	57	593	[13]
	R	ATCCAGATCCGCAAAGGTTA			
qnrB	F	ATGACGCCATTACTGTATAA	53	560	[13]
	R	GATCGCAATGTGTGAAGTTT			
qnrS	F	ATGGAAACCTACAATCATAC	55	492	[13]
	R	AAAAACACCTCGACTTAAGT			

3.Results and discussion

Bacterial growth

Data in table 2 showed out of 374(100%) specimens distributed between 199(53 %) specimens from male patients and 176 (47%) obtained from female patients, also current results revealed that 157(41.9%) of specimens was obtained as single growth, and 28(7.4%) of specimens was as mix growth while 190 (50.7%) of specimens was recorded as no bacteria growth may be due to fungal or viral infection. At same time this work recorded 155(41.33%) as gram-negative bacteria.

Table 2: Distribution of bacterial growth and non-growth in the study

Status	Female	Male	total	P-value
Single growth	61(16.3%)	96(25.6%)	157(41.9%)	0.0289*
Mix growth	15(4%)	13(3.4%)	28(7.4%)	
No growth	100 (26.7%)	90(24%)	190 (50.7%)	
Total	176 (47%)	199(53 %)	375(100%)	

Results as listed in table (3) indicate that 28(7.46%) *A. baumannii* isolates were obtained from all five clinical sources, although in differing quantities. The greatest isolation rate was seen in burn patients at 17 (4.53%), followed by 4 (1.07%) isolates from patients with respiratory tract infections, and 3 (0.8%) from blood source records. Both diabetic foot infections and urinary tract infections were recorded in 2 cases (0.53%).

Table 3: distribution of *A. baumannii* according to sex and specimens.

Source	Sex	Specimen	<i>A.baumannii</i>	Total <i>A. baumannii</i>
Urine	Female	56(14.93%)	2(0.53%)	2(0.53%)
	Male	19(5.07%)	0(0.0%)	
Sputum	Female	35(9.33%)	2(0.53%)	4(1.07%)
	Male	40(10.67%)	2(0.53%)	
Blood	Female	36(9.6%)	1(0.27%)	3(0.8%)
	Male	39(10.4%)	2(0.53%)	
Diabetic foot	Female	20(5.33%)	1(0.27%)	2(0.53%)
	Male	55(14.67%)	1(0.27%)	
Burns	Female	29(7.73%)	8(2.13%)	17(4.53%)
	Male	46(12.27%)	9(2.4%)	

Total	375(100%)	28(7.46%)	28(7.46%)
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The study showed That *A. baumannii* isolates were 28 (100%) resistance to Cefoxitine, Ceftazidime and Ciprofloxacin. While it recorded that 27(96.42%) of isolates were resistance to meropenem, while 26(92.85%) of isolates were resistance to tobramycin and ampicillin-Sulbactam, at same time (89.28%) of isolates were resistance to gentamicin antibiotic (table, 4).

Table 4: Antibacterial agent susceptibility of *A. baumannii* isolates

Antibacterial agents	Resistance (%)	Intermediate (%)	Sensitive (%)
Ampicillin-Sulbactam	26(92.85%)	2(7.14%)	0(0.0%)
Cefoxitine	28(100%)	0(0.0%)	0(0.0%)
Ceftazidime	28(100%)	0(0.0%)	0(0.0%)
Meropenem	27(96.42%)	0(0.0%)	1(3.57%)
Gentamicin	25(89.28%)	1(3.57%)	2(7.14%)
Tobramycin	26(92.85%)	0(0.0%)	2(7.14%)
Ciprofloxacin	28(100%)	0(0.0%)	0(0.0%)

Detection of quinolone resistance genes in *A. baumannii*.

Results of PCR analysis for 28 isolates of *Acinetobacter baumannii* showed that, the qnrA gene was detected in 11 (39%), were the qnrB gene was only in 7(25%) of *A. baumannii* isolates, as for the qnrS gene showed a high frequency reached 21 (75%) of the isolates.

Bacterial growth

Numerous previous investigations have shown *A. baumannii*'s ability to cause diverse infections in both men and females. A research by Alwohaili and Al-Awade [14] indicated that, among a total of 40 *A. baumannii* isolates, 25 (62.5%) derived from females and 15 (37.5%) from males. The isolates were classified

as follows: 17 (42.5%) from sputum, 10 (25%) from wounds, 7 (17.5%) from urine, 5 (12.5%) from blood, and 1 (2.5%) from fluid specimens. Khan et al. [15] write that the study included 480 individuals, of whom 365 (76%) were male and 115 (24%) were female, with a prevalence of *A. baumannii* of 14.58% in diabetic foot infections. Similarly, a study by Saleh et al. [16] they reported that the frequency of *Acinetobacter* included 55 isolates (83%) from burn cases, 32 (78%) from wounds, and 13 (92.8%), 7 (46.6%), 3 (100%), and 3 (50%) from diabetic foot ulcers, tonsils, cerebrospinal fluid, and urine, respectively. The prevalence of this bacterium was 67 (59.3%) in men and 46 (40.7%) in females. Variation in number of bacteria among studies may be due to number of specimens or geographical region of study.

Antimicrobial agents susceptibility of A. baumannii isolates

A recent study conducted in Duhok, Iraq, by Yousif et al. [17], identified 72 out of 350 *A. baumannii* isolates from various clinical specimens, including wounds, burns, sputum, blood, and urine. *A. baumannii* demonstrated resistance rates of 100% to Cefoxitin, Ciprofloxacin, and Tobramycin, with resistance to Meropenem at 79.2% and to Gentamicin at 75.0%.

This investigation confirms the results of Alwohaili et al. [14], which examined *A. baumannii* isolates in Karbala and shown significant resistance to many antibiotic classes, including cephalosporins, in diverse clinical samples. Antibiotic susceptibility evaluations conducted on 50 *A. baumannii* isolates revealed resistance rates of 100% to ampicillin-sulbactam and 96.0% to ceftazidime. Furthermore, 98.0% of isolates exhibited resistance to ciprofloxacin, while the resistance rate to tobramycin was 68% [18].

Antibiotic resistance may result from diminished membrane permeability, increased antibiotic efflux, obstructed target access, or the hydrolysis or modification that directly inactivates medications [19]. *A. baumannii* may develop one or more pathways that might lead to resistance to all commonly used antibiotics under certain situations.

Molecular screening of virulence genes in A. baumannii isolates

The current investigation revealed that the prevalence of the *qnrA* gene in *A. baumannii* isolates was 39%. This finding is inferior to that reported in an Iraqi study conducted by Mshachal et al. [20], which indicated a *qnrA* gene prevalence of 60.6%. A similar result was observed in another study conducted in Baghdad, where 44 isolates of *A. baumannii* collected from various clinical samples exhibited a *qnrA* gene prevalence of 73.3%. The *qnrB* gene was identified in 25% of *A. baumannii* isolates, reflecting a comparatively lower prevalence than *qnrS*. This finding is less than the 73.3% reported by Jaber et al. [21], in a study conducted in Baghdad City, yet it surpasses the 11% detected by Ali et al. [22] in Suez Canal hospitals.

In 2022, Turkey collected 29 *A. baumannii* isolates from various clinical sources, revealing a significantly elevated prevalence of the *qnrB* gene at 82.8% [23]. The *qnrS* gene exhibited the highest prevalence (75%) among fluoroquinolone resistance genes in *A. baumannii* isolates in the present study. Conversely, a study by Moosavian et al. [24] reported a *qnrS* distribution of 3.2%, while another investigation of *A. baumannii* isolates from various hospitals indicated a significantly lower prevalence of the *qnrS* gene at 0.3% [23]. In a separate study, the *qnrS* gene was not identified in any of the 60 *A. baumannii* isolates [25].

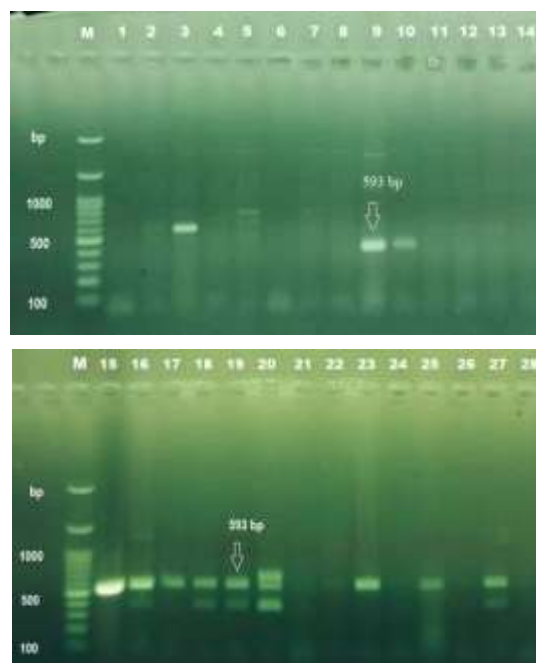


Fig 1: Frequency of *qnrA* gene among 28 isolates of *A. baumannii*.

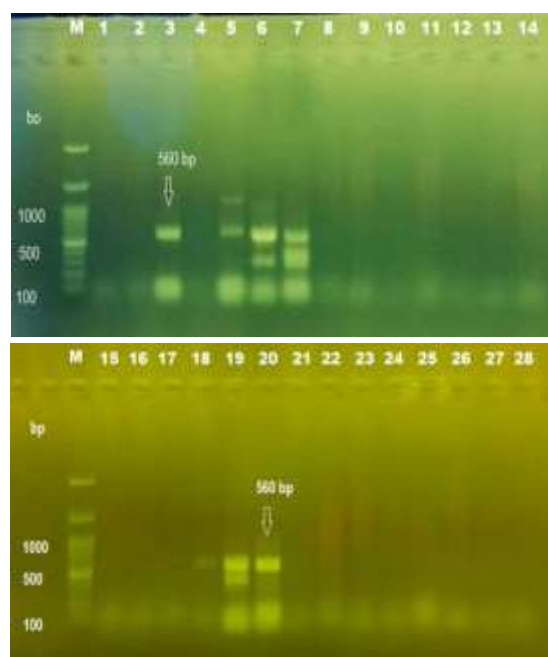


Fig 2: Frequency of qnrB gene among 28 isolates of *A. baumannii*

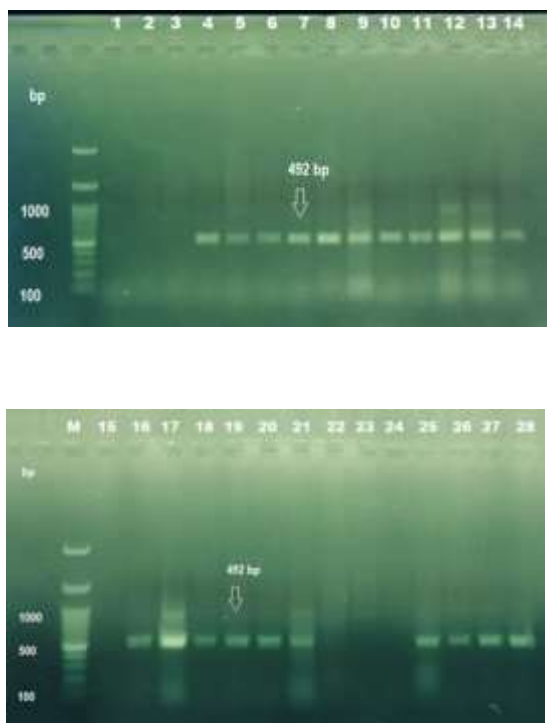


Fig 3: Frequency of qnrS gene among 28 isolates of *A. baumannii*

Conclusion

Data revealed the frequency of *A. baumannii* in several human infections as well as its ability to resist different antibiotics. The scarcity of efficacious antibiotics for the treatment of *A. baumannii* infections is concerning. The findings underscore the need for ongoing surveillance and prudent antibiotic management.

Acknowledgement

We would like to thank our Al-Sadr Hospital, Al-Najaf Al-Ashraf Teaching Hospital, Al-Zahraa Teaching Hospital for Maternity and Pediatrics as well as some chief clinical laboratories and the patients for their participation in this study

Funding Information

None

Author's Contributions

All authors had equal contribution to this manuscript

Ethics

This study was conducted under approval by the medical ethics committee at University of Kufa (2025).

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