Microbes and Infectious Diseases 2025; 6(4): 6668-6676

Microbes and Infectious Diseases

Journal homepage: https://mid.journals.ekb.eg/

Original article

Incidence of resistance pattern and biofilm-forming genes of *Acinetobacter baumannii* isolated from Iraqi children

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ARTICLE INFO

Article history:
Received 28 December 2024
Received in revised form 9 January 2025
Accepted 14 January 2025

Keywords:
A. baumannii
Biofilm
Antibacterial susceptibility
multidrug resistant
*Omp*A gene.

ABSTRACT

Background: *Acinetobacter baumannii* is frequently implicated in nosocomial infections, causing a diverse range of illnesses in humankind. The objective of the study was to investigate the multidrug-resistant and biofilm-forming *A. baumannii* isolated from Iraqi children. **Methods:** Seventy-five blood and urine samples were collected from children in Baghdad, Iraq. VITEK 2 compact was used for identification and antibiotic susceptibility testing. Biofilm production was assessed using microtiter plates, and biofilm genes such as *csuE, pgaB, bfmS,* and *ompA* were detected using conventional PCR. **Results:** Of the sample size, 54 isolates were identified as *A. baumannii*. Among the tested antibiotics, ticarcillin (68.5%) showed the highest resistance rate, while colistin showed the highest susceptibility rate (92.6%). Furthermore, 61.11% of the isolates were XDR, and 29.6% were MDR. All the isolates showed the capability for biofilm production; 63% and 37% of isolates produced robust and moderate biofilm, respectively. All the isolates were determined to contain a minimum of one gene relevant to biofilm formation; however, the most prevalent gene was *csu*E (100%). **Conclusion:** High resistance rates were shown among *A. baumannii* strains. Moreover, biofilm gene *csu*E was shown in all the strains. However, further experimentation to evaluate biofilms' involvement in the pathophysiology of *A. baumannii* infections is mandatory.

Introduction

Acinetobacter baumannii is an aggressive microbe that belongs to the category of non-fermentative Gram-negative bacteria. *A. baumannii* has been identified as a significant infection in healthcare settings, leading to infections associated with higher patient morbidity rates, death, and treatment expenses [1]. This emergence has been observed throughout the past few decades. *A. baumannii* is frequently encountered within healthcare facilities and is responsible for various healthcare-associated infections, including blood vessels, UTIs, meningitis, and wound infections [2,3].

The clinical importance of this organism has been driven by its exceptional capacity to increase the expression or acquire resistance genes, posing a significant danger to the effectiveness of current antibiotics. This array of resistance mechanisms has enabled the organism to resist

DOI: 10.21608/MID.2025.348347.2417

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various antibiotics commonly found in hospital environments. *A. baumannii* can endure in humid and dry environments for extended periods, potentially spanning many weeks or months. Consequently, this microorganism is prevalent in several settings, including natural environments, healthcare facilities, human skin, and membranes [4]. The escalating risk of *A. baumannii* is connected with a substantial percentage of strains exhibiting resistance to antimicrobial agents. *A. baumannii* belongs to a bacterial group responsible for nonsocial antibacterial resistance, referred to as ESKAPE, which includes *Enterococcus faecium*, *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Enterobacter* spp [5].

Developing nations, such as Iraq, primarily face the challenge of resistant bacteria due to their inadequate medical resources. Several multidrug resistance (MDR) instances have been documented in Iraq [6-8]. The ability of MDR *A. baumannii* to thrive and survive in different healthcare atmospheres and under arid conditions has established it as a significant contributor to nosocomial illness on a global scale [9]. One significant aspect contributing to long-term and ongoing infections and the development of antibiotic resistance in *A. baumannii* is its aptitude for inhabiting and establishing biofilm on both living and non-living surfaces [10]. The rate of biofilm development in *A. baumannii* is reported to range from 80% to 91%, which is significantly high compared to other species [4]. Prior research has documented a correlation between the creation of biofilms and the increase of antibacterial resistance in *A. baumannii* isolates [11].

Various virulence factors are implicated in the biofilm formation of *A. baumannii*. These factors include chaperon-usher pilus (Csu), quorum sensing system, biofilm-associated protein (Bap), extracellular exopolysaccharide (EPS), outer membrane protein A (OmpA), poly-β-(1,6)-N-acetyl glucosamine (PNAG), and two-component system (BfmS/BfmR) [10,12].

OmpA assumes a significant function in attaching to and invading epithelial cells through its interaction with fibronectin. The protein in concern is also implicated in several biological processes, such as serum resistance, biofilm formation and antibiotic resistance [13,14]. The CsuA/BABCDE complex is pivotal in the origination of biofilm on abiotic surfaces. Earlier investigations have proved that silencing the *csu*E gene stops pilus assembly and inhibits the biofilm [9,11]. The transcriptional activity of the csu operon is controlled by a two-module regulatory mechanism known as bfmRS. Based on earlier studies, it has been observed that inactivation of the *bfm*S gene leads to a reduction in biofilm production in *A. baumannii* [15].

Our comprehensive assessment showed that limited research had been conducted on antibiotic susceptibility patterns and biofilm-forming *A. baumannii* isolated from Iraqi children. Therefore, the investigation aimed to study the frequency of multidrug resistance and analyze the phenotypic and genotypic biofilm formation properties of *A. baumannii* in Iraqi children.

Material and Methods

Sample collection

Seventy-five blood and urine samples were collected from 10-year-old children who came into the Central Teaching Hospital of Pediatrics in Baghdad, Iraq, from August to December 2022. The rationale for this experiment was comprehensively deliberated and elucidated to the selected children's parents, and an informed consent form was gained. The ethical approval was obtained from the Central Teaching Hospital of Pediatrics, Baghdad, Iraq.

Bacterial identification

The specimens were subjected to inoculation on blood agar and MacConkey agar (HiMedia, India), followed by incubation at 37°C for 18 h. Colony morphology was presented as yellow to pink on MacConkey agar. While on blood agar, colonies appeared white to creamy, with smooth and transparent colonies, without any hemolysis or pigmentation. The suspected colonies were further confirmed as *A. baumannii* by the VITEK 2 compact system using a GN detection kit (bioMérieux, France).

Antibacterial susceptibility

The VITEK 2 compact system was employed to determine antibacterial susceptibility (bioMérieux, France) using an AST-GNB kit as described by [7], which included a susceptibility test for 14 different antibiotics belonging to diverse classes of antibiotics. The antibiotics involved in this study were ticarcillin, ticarcillin/clavulanic acid, piperacillin, piperacillin/ tazobactam, ceftazidime, cefepime, imipenem, meropenem, gentamicin, tobramycin, ciprofloxacin, minocycline, colistin and trimethoprim/sulfamethoxazole. The MDR isolates were characterized as being resistant to a minimum of one compound in three or more distinct drug classes. Extensively drug-resistant (XDR) bacteria demonstrate resistance to a minimum of one agent within each antibacterial class, with the exception of two or fewer agents [2].

Biofilm production assay

A microtiter plate technique was used to assess the biofilm-forming *A. baumannii* isolates [11]. Briefly, the isolates were cultured for one night in tryptic soy broth supplemented with 0.25% glucose (Himedia, India). The cultivation process was carried out at a temperature of 37 °C. The removal of free cells was followed by a triple wash of the biofilms using sterile phosphate-buffered saline and subsequent fixation using 150 mL of 99% (v/v) methanol (Merck & Co.). The wells were treated with a 1% (w/v) solution of crystal violet for 20 minutes at room temperature. The crystal violet dye was dissolved in a solution consisting of 33% (v/v) ethanol and acetone in a ratio of 80:20 (v/v) for 20 minutes. Subsequently, the absorbance of the resulting solution was determined at an optical density (OD) of 595 nm. The recording of biofilm formation was conducted using the following criteria: no biofilm formation (A595 < 1); weak biofilm formation (1 < A595 ≤ 2); moderate biofilm formation (2 < A595 ≤ 3); and strong biofilm formation (A595 > 3). The reported values represent the average of three readings.

Detection of biofilm-related genes

The genomic DNA extraction kit (Geneaid Biotech Ltd, Taiwan) was employed to extract the bacterial genome as per the manufacturer's directions. The concentration of DNA was assessed by quantifying the absorbance of the sample at 260 nm. Conventional PCR was conducted to identify and detect biofilm-linked genes: *csu*E, *pga*B, *bfm*S, and *omp*A. The primers and the annealing temperature are presented in Table 1. The sequences were compared with the NCBI BLAST website database (http//www.ncbi.nlm.nih.gov/blast) to identify the genes. The GenBank accession numbers of the genes were acquired.

Statistical analysis

The study employed the Statistical Analysis Software (SAS, 2018) to determine the impact of various factors on the research variables. A chi-square test assessed the statistical significance of the data. A *p*-value of < 0.05 was deemed significant.

Results

Out of 75 collected samples, 54 isolates were identified as *A. baumannii*. Of the 54 *A. baumannii* isolates, 46 (85.2%) were recovered from blood and 8 (14.8%) from urine (P value ≤0.01).

Among the tested isolates, 33 (61.11%) were shown to be XDR, 16 (29.6%) were MDR, and 5 (9.2%) were entirely susceptible. The highest level of resistance was observed in ticarcillin (68.5%), followed by ceftazidime (59.3%), piperacillin (57.4%), and ticarcillin/clavulanic acid (55.6%). In contrast, colistin exhibited the highest susceptibility rate (92.6%), followed by tobramycin (66.7%), minocycline (66.7%), and trimethoxazole (64.8%). The pattern of antibiotic susceptibility of *A. baumannii* is demonstrated in Table 2.

All the isolates of *A. baumannii* demonstrated the capability to generate biofilms. All isolates of *A. baumannii* were found to possess at least one gene associated with biofilm formation. The gene with the highest frequency was *csu*E, observed in all strains (100%), while *pga*B, *omp*A, and *bfm*S were present in 95%, 95%, and 90% of the strains, respectively. The molecular size of the biofilm genes *csu*E, *pga*B, *bfm*S, and *omp*A were 228 bp, 366 bp, 455 bp, and 578 bp, respectively, as shown in Figure 1. The GenBank accession numbers of *csu*E, *pga*B, *bfm*S, and *omp*A genes were listed in table 3

Table 1. The primer sequences used and the product sizes obtained for each gene.

Gene	Primer Sequence (5ˈ-3ˈ)	Product Size(bp)	Annealing	References
***csu*E**	CGGGCACAGAATAGAACCAT (Forward)	228 bp	57	This study
	ATGCTCAGACCGGAGAAAAA (Reverse)			
***pga*B**	GCGCTGTGAATGAGCAATTA (Forward)	366 bp	56	This study
	CAATGTCATACTGCCCATCG (Reverse)			
***bfm*S**	TGAAATCGGGTGATTTGTCA (Forward)	455 bp	57	This study
	AAGGTAACGACGCTCTGCAT (Reverse)			
***omp*A**	GTTAAAGGCGACGTAGACG (Forward)	578 bp	56	This study

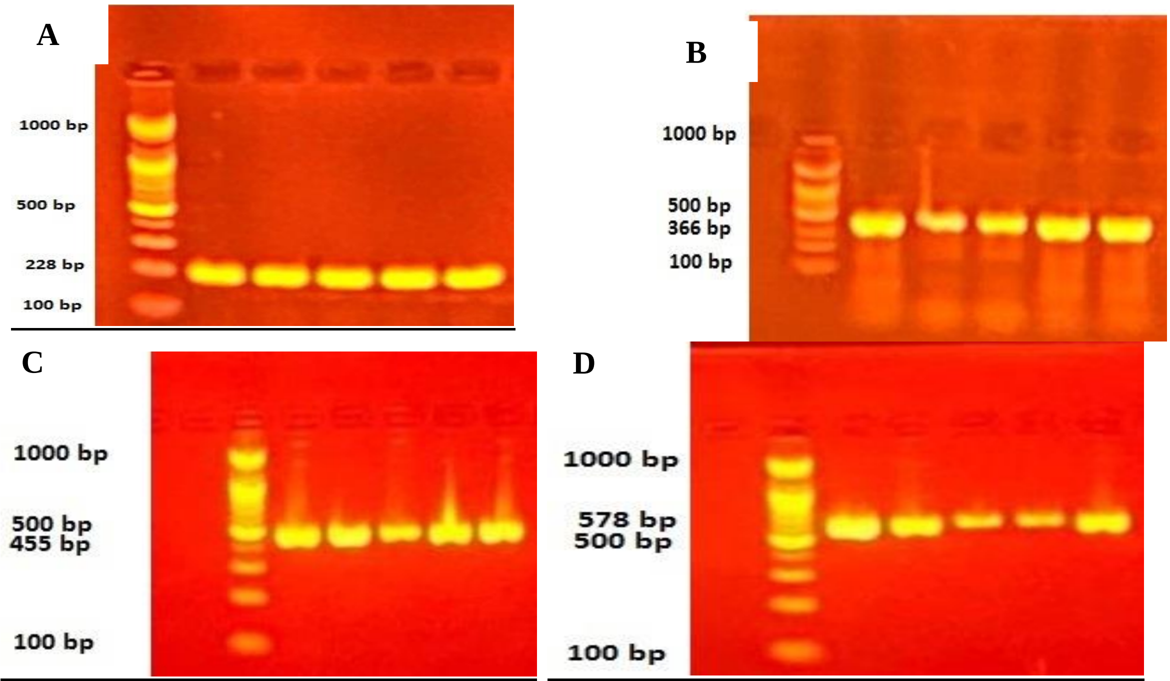
Table 2. Antibacterial susceptibility profile of *A. baumannii*

Antibiotic	54 isolates		*P* value
	Resistant (%)	Sensitive (%)	
Ticarcillin	37 (68.5%)	17 (32.5%)	0.0006
Ceftazidime	32 (59.3%)	22 (40.7%)	0.0084
Piperacillin	31 (57.4%)	23 (42.6%)	0.028
Ticarcillin / Calvulanic acid	30 (55.6%)	24 (44.4%)	0.092
Cefepime	29 (53.7%)	25 (46.3%)	0.237
Piperacillin/ Tazobactam	27 (50%)	27 (50%)	1.00
Ciprofloxacin	26 (48.1%)	28 (51.9%)	0.855
Gentamicin	25 (46.3%)	29 (53.7%)	0.237
Meropenem	24 (44.4%)	30 (55.6%)	0.092
Imipenem	24 (44.4%)	30 (55.6%)	0.092
Trimethoprim/Sulfamethoxazole	19 (35.2%)	35 (64.8%)	0.0096
Tobramycin	18 (33.3%)	36 (66.7%)	0.0071
Minocycline	18 (33.3%)	36 (66.7%)	0.0071
Colistin	4 (7.4%)	50 (92.6%)	0.0001
P-value	0.0001 **	0.0001 **	

Table 3. GenBank accession numbers of the genes.

Sl No.	Seq No.	Sample No.	Gene	Accession number
1.	Seq 1	ABFH1	*bfm*S	PP855477
2.	Seq 2	ABFH2	*bfm*S	PP855478
3.	Seq 3	ABFH3	*bfm*S	PP855479
4.	Seq 4	ABFH4	*bfm*S	PP855480
5.	Seq 5	ABFH5	*bfm*S	PP855481
6.	Seq 6	ABFH1	*omp*A	PP855482
7.	Seq 7	ABFH2	*omp*A	PP855483
8.	Seq 8	ABFH3	*omp*A	PP855484
9.	Seq 9	ABFH4	*omp*A	PP855485
10.	Seq 10	ABFH5	*omp*A	PP855486
11.	Seq 11	ABFH1	*csu*E	PP855487
12.	Seq 12	ABFH2	*csu*E	PP855488
13.	Seq 13	ABFH3	*csu*E	PP855489
14.	Seq 14	ABFH4	*csu*E	PP855490
15.	Seq 15	ABFH5	*csu*E	PP855491
16.	Seq 16	ABFH1	*pga*B	PP855492
17.	Seq 17	ABFH2	*pga*B	PP855493
18.	Seq 18	ABFH3	*pga*B	PP855494
19.	Seq 19	ABFH4	*pga*B	PP855495
20.	Seq 20	ABFH5	*pga*B	PP855496

Figure 1. PCR bands of the biofilm genes of *A. baumannii* isolates. **A:** Detection of *csu*E gene (228 bp). **B:** Detection of *pga*B gene (366 bp). **C:** Detection of *bfm*S gene (455 bp). **D:** Detection of *omp*A gene (578 bp).



Discussion

The aptitude of MDR *A. baumannii* to persist and endure in diverse hospital sites has made it a significant contributor to nosocomial infections on a global scale [16]. The effective management of MDR *A. baumannii* presents considerable issues in various Asian countries [9,11,17]. The present study revealed 61.11% of the isolates exhibited XDR, while 29.6% displayed MDR. These findings were supported by previous research conducted in Iran, where 32% of the screened strains were resistant to all drugs, and 91% were classified as XDR [11]. In Jordan, 478 *A. baumannii* isolates were identified as MDR, accounting for 78.8% of the reported cases [1]. Based on a prior study conducted in Iraq, it was shown that the incidence of MDR *A. baumannii* isolates was observed to be 100% [17].

The present investigation provides evidence that colistin exhibits the lowest resistance rate against *A. baumannii*, which concurs with findings from recent reports in Iraq, Iran, Jordan, and India [1,2,17,18]. According to Depka et al., colistin is the predominant therapeutic approach for *A. baumannii* infections [19]. This finding correlates with the outcomes of our investigation.

AL-Kadmy et al., displayed that all isolates of *A. baumannii* exhibited resistance to trimethoprim/sulfamethoxazole and ciprofloxacin [17]. In contrast, the current study presented that 35.2% and 48.1% of the isolates revealed resistance to trimethoprim/sulfamethoxazole and ciprofloxacin, respectively. According to a recent study, it was found that over 90% of the isolates of *A. baumannii* displayed resistance to ticarcillin/clavulanic acid, piperacillin/tazobactam, ceftazidime, ciprofloxacin, imipenem, and meropenem [20]. In comparison, our results showed less resistance to ticarcillin/clavulanic acid (55.6%), piperacillin/ tazobactam (50%), ceftazidime (59.3%), ciprofloxacin (48.1%), and 44.4% to both imipenem and meropenem (Table 2).

Vázquez-López et al., reported that resistance to carbapenems is frequently associated with reduced sensitivity to other classes of antibiotics, such as fluoroquinolones [3]. Depka et al., detected that all carbapenem-resistant *A. baumannii* strains were resistant to fluoroquinolones [19]. These findings are consistent with our research results, where 44.4% and 48.1% of *A. baumannii* isolates revealed resistance to meropenem and imipenem, and ciprofloxacin, respectively (Table 2).

The potential of *A. baumannii* to colonize and establish biofilm on both living and non-living surfaces is a significant element contributing to the development of chronic and persistent infections [21]. Based on the outcomes found, it was observed that all isolates of *A. baumannii* showed the capacity to generate biofilm. Furthermore, 63% of the isolates exhibited an aptitude for biofilm production. Our result in this regard was comparable to those reported in the earlier investigations, which showed that between 53% and 75% of A. baumannii isolates form biofilms [11,12]. Previous research has documented a favorable association between the creation of biofilms and the emergence of antibacterial resistance in isolates of *A. baumannii* [22]. The current study observed that all *A. baumannii* isolates that exhibited a high capacity for biofilm formation were classified as XDR. Moreover, biofilm production in susceptible isolates was more potent than in MDR and XDR [23]. Therefore, further investigation is necessary to comprehensively comprehend the correlations between biofilm formation and the emergence of resistance.

On the other hand, numerous investigations have provided evidence indicating that biofilm-associated genes of *A. baumannii*, such as *csu*E, *omp*A, *bap*, *eps*A, and *bfm*S, played a considerable influence in both biofilm formation and the development of antibacterial resistance [4,11,22]. Based on the findings of our study, it was indicated that the *csu*E gene exhibited the highest prevalence rate of 100%. Subsequently, the *pga*B and *omp*A genes were found to have prevalence rates of 95% each, while the *bfm*S gene demonstrated a prevalence rate of 90%. Zeighami et al., revealed that 100% of the antibiotic-resistant *A. baumannii* isolates were capable of generating moderate or vigorous biofilm and were detected to have biofilm genes at high rates, i.e, *csu*E (100%), *pga*B (98%), *eps*A (95%), *ptk* (92%), *bfm*S (92%), and *omp*A (81%) [11]. The gene *csu*E is a component of the usher-chaperone assembly system, which plays a role in facilitating attachment and promoting biofilm development. The *csu*E gene constituted 100% of the isolates examined in this investigation. This is comparable with the previous study, where the *Csu*E gene was found in 91.6% of *A. baumannii* isolates [24].

Wie et al., showed that 100% of the isolates were carrying BfmRS, which indicated the motility of *A. baumannii* [25]. Furthermore, Palethorpe et al., reported that BfmRS regulates type IV pili development and the manufacture of siderophores. A study also demonstrated that the BfmS sensor kinase functions as a BfmR phosphatase, negatively regulating BfmR activity [15]. The latest research displayed that BfmS and BfmR are responsible for the motility of *A. baumannii* based on light [26]. The present study indicated a high prevalence of the *bfm*S gene (90%) among the isolated *A. baumannii*.

The isolates exhibiting the highest level of antibiotic resistance were found to possess numerous genes linked to biofilm development. The OmpA protein of *A. baumannii* is likely crucial for its ability to attach to human epithelial cells, facilitate the formation of biofilms, and confer resistance to antimicrobial agents [27]. Our investigation revealed a significant prevalence of the *Omp*A gene, with a frequency of 95%. Recent studies showed the importance of OmpA protein as a potential virulence factor in *A. baumannii*, which is responsible for cell death in macrophages and lung epithelium [28,29].

Conclusion

The investigation highlights the importance of XDR and MDR *A. baumannii*, as all the isolates were obtained from children. The investigation indicated a significant occurrence of biofilm-forming XDR *A. baumannii*, characterized by a notable occurrence of biofilm-associated genes *csu*E, *pga*B, *bfm*S, and *omp*A. Effective methods of monitoring and controlling activities are crucial to limit the growth and spread of XDR *A. baumannii*. The study emphasizes the importance of conducting susceptibility testing before prescribing antibiotics to reduce future antibiotic resistance. Additional investigations into the genetic aspects will enhance our comprehension and yield innovative perspectives in developing treatments and preventive measures against *A. baumannii* biofilm-associated illnesses.

Conflicts of Interest

The authors declare no conflict of interest.

Financial disclosure

No financial support were received.

Data availability

Data available on request / reasonable request.

Authors' contribution

Each author made an equal contribution to the production of the initial draft, and they have all viewed and approved the manuscript.

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