Iranian Journal of Microbiology

Volume 17 Number 4 (August 2025) 549-558
DOI: http://doi.org/10.18502/ijm.v17i4.19228

ORIGINAL ARTICLE

Exploring the genetic diversity and the association of drug resistance and biofilm production in *Acinetobacter baumannii* strains isolated from burn wound infections

Sanaz Khashei[1], Hossein Fazeli[1*], Fateh Rahimi[2*], Vajihe Karbasizade[1]

[1]Department of Microbiology, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran
[2]Department of Cell and Molecular Biology & Microbiology, Faculty of Biological Science and Technology, University of Isfahan, Isfahan, Iran

Received: December 2024**, Accepted:** June 2025

ABSTRACT

Background and Objectives: *Acinetobacter baumannii* is considered a troublesome cause of infection in burn units, where its capability to form biofilm and resist antibiotics significantly hampers therapeutic success. This study explored the correlations between antimicrobial resistance profiles, biofilm-producing capacity, and genetic diversity of *A. baumannii* strains from patients with burn wound infection in Isfahan, Iran.

Materials and Methods: Ninety-six isolates were analyzed for antibiotic resistance using the disk diffusion technique and for biofilm formation through the microtiter dish assay. The prevalence of ten biofilm-related genes was investigated using specific primers. Clonal relatedness among bacterial strains was defined by Enterobacterial Repetitive Intergenic Consensus-Polymerase Chain Reaction (ERIC-PCR).

Results: A vast majority of isolates (99%) exhibited resistance to meropenem, ciprofloxacin, ceftriaxone, cefotaxime, piperacillin-tazobactam, and imipenem, qualifying them as extensively drug-resistant (XDR). Twenty-five percent of the strains were strong biofilm formers, while 68% demonstrated moderate or weak biofilm formation. The most commonly identified biofilm-related genes included *bfmR* (100%), *ompA* (100%), and *bap* (99%). A significant association was found between the production of biofilm, resistance to aminoglycosides, and the presence of *csuE* and *bap* genes. ERIC-PCR typing showed the presence of 3 clonal types and 7 single types, with biofilm producers predominantly clustering to clonal type 2.

Conclusion: This work highlights a notable prevalence of biofilm-producing XDR *A. baumannii* in burn patients, underscoring the need for continuous surveillance and enhanced infection control strategies.

Keywords: Burns; *Acinetobacter baumannii*; Drug resistance; Biofilm; Molecular typing

INTRODUCTION

Burn wound infections lead to a considerable mortality rate (33 to 80%) due to the development of life-threatening conditions like bacteremia and sepsis (1). *Acinetobacter baumannii*, particularly its drug-resistant forms, have emerged as one of the most concerning pathogens in hospitals and is linked with post-burn infections (2). The emergence and growing prevalence of extensively drug-resistant

*Corresponding authors: Hossein Fazeli, Ph.D, Department of Microbiology, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran. Tel: +98-3137929039 Email: h_fazeli@med.mui.ac.ir

Fateh Rahimi, Ph.D, Department of Cell and Molecular Biology & Microbiology, Faculty of Biological Science and Technology, University of Isfahan, Isfahan, Iran. Tel: +98-3137932471 Fax: +98-3137932456 Email: f.rahimi@sci.ui.ac.ir

(XDR) strains have significantly narrowed therapeutic choices and represent a major concern for nosocomial infection control. One of the key contributors to its resistance is the organism's capacity for robust biofilm formation, which plays a pivotal role in its ability to withstand antimicrobial agents' wide range of virulence determinants involved in the sequential stages of biofilm development (3). The outer membrane protein A (OmpA) is the most abundant porin associated with drug resistance, contributing to antimicrobial resistance, adherence to epithelial tissues, and biofilm production on living surfaces by mediating porin-fibronectin interactions. The biofilm-associated protein, which is encoded by the *bap* gene has a fundamental role in the early adhesion phase and subsequent maturation of the biofilm. The *pgaABCD* locus controls the synthesis of poly-β-(1,6)-N-acetyl glucosamine as a main building block in *A. baumannii*'s biofilm (4). Moreover, the maturation of biofilm is facilitated by the chaperone-usher pilus system which is encoded by the *csu* operon. Disruption of the *csuE* gene, which encodes a presumed tip adhesin, leads to a failure in pilus formation and consequently impairs biofilm development (5). The expression of the *csu* operon is controlled by the *bfmRS* genes (6). In addition, the production of N-hydroxy dodecanoyl-L-homoserine lactone, synthesized by the autoinducer synthase encoded by the *abaI* gene influences the later phases of biofilm production via the quorum sensing process (4). The RND (resistance-nodulation-division) efflux pump family is responsible for much of the intrinsic drug resistance due to their extremely broad substrate specificity in Gram-negative pathogens. Moreover, the transformation from planktonic to biofilm states in *A. baumannii* is influenced by exporting quorum-sensing molecules through RND efflux pumps (7). Among these, three major RND-type efflux systems, known as the Ade systems, have been identified, with the genes *adeB, adeG,* and *adeJ* serving as essential components of these mechanisms (8). The epidemiological study of *A. baumannii* clones will provide a critical aid for controlling the spread of antibiotic-resistant strains, optimizing antimicrobial therapies, decreasing treatment-related costs, and revealing the mode of pathogenicity. Despite the epidemiological importance of *A. baumannii,* the exact role of its virulence factors in antibiotic resistance and biofilm production is not well understood in burn wound infections. Therefore, this study aimed to investigate the relationship between the production of biofilm and the genetic variability of XDR *A. baumannii* strains isolated from burn patients.

MATERIALS AND METHODS

Collection and characterization of *A. baumannii* isolates. This cross-sectional study was carried out on *A. baumannii* isolates obtained from burn wound samples of hospitalized patients at Imam Musa Kazim Burn Hospital in Isfahan, Iran, over a 10-month period (February to November 2022). Based on a previously reported biofilm formation prevalence of 90% among *A. baumannii* isolates (9), the required sample size for this study was estimated to be approximately 96, using the following formula (10):

$n = (p(1-p)\ z^2) / d^2$

In this formula, *n* represents the required sample size, *p* is the prevalence of biofilm production (90%), and *z* and *d* are 1.96 and 0.05, respectively.

The collected isolates were preliminarily identified using morphological, physiological, and standard biochemical tests (11, 12). The molecular confirmation of these isolates as *A. baumannii* was done through PCR for the $bla_{\text{OXA-51-like}}$ gene, as described by Falah et al. (13).

Determination of antibiotic resistance profile. Resistance of bacterial strains against 13 different antibiotics was assessed using the Kirby-Bauer technique (14). For this purpose, the effect of antibiotics belonging to six different categories including ampicillin-sulbactam and piperacillin-tazobactam (β-lactam/β-lactamase inhibitor combinations); ceftazidime, ceftriaxone, and cefotaxime (cephalosporins); meropenem and imipenem (carbapenems); gentamicin and amikacin (aminoglycosides); levofloxacin and ciprofloxacin (fluoroquinolones); and tetracycline and doxycycline (tetracyclines), was tested. The classification of strains into multidrug-resistant (MDR) and XDR categories was as follows: MDR strains exhibit non-susceptibility to at least one drug in three or more classes of antibiotics, whereas XDR strains are resisted to all but two or fewer classes of antibiotics (15).

Determination of biofilm production ability. The capacity of the production biofilm was evaluated via a quantitative microtiter plate (MTP) assay (16). Briefly, 100 µL of bacterial suspensions, with an optical density of 600 nm (OD_{600}) equal to 0. 1, were added to the wells of a 96-well plate containing 100 µL of

TSB supplemented with 10% bovine serum albumin (BSA). Following 24 hours of incubation at 37°C, plates were gently rinsed with physiological serum (Sodium chloride 0.9%). Then, 200 µL of 96% ethyl alcohol was added to wells and incubated at 25°C for 15 minutes. Following staining for 15 minutes with 1% (w/v) crystal violet, the wells were rinsed with physiological serum, and the dye bound to the biofilm was solubilized using 30% (v/v) acetic acid. The OD values were read at 570 nm using an ELISA reader. The capability of each strain to produce biofilm was scored as strong (ODi > 4ODc), moderate (2ODc < ODi ≤ 4ODc), weak (ODc < ODi ≤ 2ODc), and negative (ODi ≤ ODc).

The ODc represents the mean OD of the negative control, and ODi refers to the OD of the test strains (17). Wells without bacterial inoculation and the type strain *A. baumannii* ATCC 19606 were considered as negative control and positive control, respectively.

Amplification of genes related to biofilm production. The prevalence of 10 biofilm-associated genes (*adeJ, bap, csuE, pgaA, abaI, bfmS, adeB, bfmR, adeG,* and *ompA*) among *A. baumannii* strains was determined using separate PCR reactions with target-specific primers which had previously been reported (16, 18).

Determination of genetic diversity. To evaluate the homology and clonal relationship of *A. baumannii*, several anonymous regions of the genome were amplified with the primers of ERIC-PCR to obtain strain-specific banding patterns (13). Strains with different bands in the ERIC banding pattern were selected and analyzed by GelJ v. 2.0 software. *A. baumannii* strains were considered to be clonally related if their ERIC-PCR patterns were ≥ 97% similar.

Statistical analysis. Independent sample t-tests and Fisher's exact test were applied to quantify the differences between the means (GraphPad Prism version 9.0). If the p-value ≤ 0.05, the result is trumpeted as statistically significant.

RESULTS

Identification of bacteria and antibiotic susceptibility testing. A total of 96 clinical isolates of *A. baumannii* were included in the present study, all of which were confirmed by PCR targeting the $bla_{\text{OXA-51-like}}$ gene. Based on the antibiotic resistance testing results the highest resistance levels were against ceftazidime, imipenem, cefotaxime, ceftriaxone, piperacillin-tazobactam, and ciprofloxacin (n=95; 99%). In contrast, the highest antibiotic susceptibility rate was observed towards gentamicin (42%, n = 40), amikacin (36%, n = 38), and ampicillin-sulbactam (32%, n = 34) (Fig. 1).

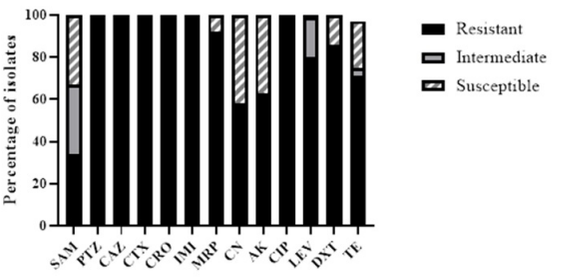

Fig. 1. Antimicrobial resistance rate among *A. baumannii* strains.

Overall, 99% (n = 95) of the strains were categorized as XDR, and only one strain represented susceptibility to all antibiotics tested. Furthermore, antimicrobial susceptibility testing revealed 13 distinct antibiotic resistance patterns among the isolates (Table 2). Moreover, one strain that was susceptible to all tested antibiotics is not included in Table 1.

Evaluation of the ability of biofilm production. Based on the results of quantitative biofilm production using MTP assay, the ODc value was set at 0.145 for the negative control. Based on this criterion, 25% (n = 24, ODi ranged from 0.7-1.647), 56% (n = 54, ODi ranged: 0.44-0.612), 11% (n = 11, ODi ranged: 0.214-0.315), and 7% (n = 7, ODi ranged: 0.088-0.132) were considered as strong, medium, weak, and non-biofilm producers, respectively.

Relationship between the level of antibiotic susceptibility and biofilm production. Statistical analysis assessing the association between the biofilm formation capacity and antibiotic susceptibility profiles revealed no significant differences in the formation of biofilm between susceptible and resistant strains for carbapenems, fluoroquinolones, penicillin/β-lactamase inhibitor combinations, tetracyclines, and cephalosporins (p > 0.05; Figs. 2a-d and f). However, a significant association was observed for aminoglycosides, where resistant strains exhibited significantly higher biofilm production (OD570) compared to susceptible ones (p < 0.05; Fig. 2e).

Table 1. The resistance profiles of *A. baumannii* strains were evaluated against a panel of antibiotics (n = 95).

Pattern	Resistance pattern	N (%)
Ten antibiotics		
1	SAM, PTZ, CTX, CRO, IMI, MRP, CN, CIP, LEV, TE	2 (2%)
2	PTZ, CAZ, CTX, CRO, IMI, AK, CIP, LEV, TE, DXT	6 (6%)
3	PTZ, CAZ, CTX, CRO, IMI, MRP, CN, AK, CIP, LEV	4 (4%)
Eleven antibiotics		
4	PTZ, SAM, CTX, CAZ, CRO, IMI, MRP, CN, AK, CIP, LEV	2 (2%)
5	SAM, PTZ, CAZ, CTX, CRO, IMI, MRP, CIP, LEV, TE, DXT	31 (33%)
6	PTZ, CAZ, CTX, CRO, IMI, MRP, CN, CIP, LEV, TE, DXT	2 (2%)
7	PTZ, CAZ, CTX, CRO, IMI, MRP, AK, CN, CIP, LEV, DXT	2 (2%)
8	PTZ, CAZ, CTX, CRO, IMI, MRP, AK, CN, CIP, LEV, TE	4 (4%)
Twelve antibiotics		
9	PTZ, CAZ, CTX, CRO, IMI, MRP, AK, CN, CIP, LEV, TE, DXT	12 (13%)
10	PTZ, SAM, CRO, CTX, IMI, CAZ, MRP, AK, CN, CIP, LEV, TE	12 (13%)
11	CAZ, SAM, CTX, CRO, MRP, AK, IMI, CIP, LEV, DXT, TE, PTZ	2 (2%)
12	IMI, PTZ, CAZ, CRO, CN, CIP, LEV, DXT, TE SAM, CTX, AK	2 (2%)
Thirteen antibiotics		
13	CTX, PTZ, CRO, MRP, AK, CN, CIP, LEV, DXT, TE, SAM, IMI, CAZ	14 (15%)

The abbreviations used are as follows: PTZ: Piperacillin-Tazobactam; TE: Tetracycline; SAM: Ampicillin-Sulbactam; IMI: Imipenem; CAZ: Ceftazidime; AK: Amikacin; CTX: Cefotaxime; MRP: Meropenem; CRO: Ceftriaxone; CN: Gentamicin; CIP: Ciprofloxacin; LEV: Levofloxacin; DXT: Doxycycline.

Relationship between the production of biofilm and the biofilm-associated genes. The most prevalent genes among all tested strains were *ompA* and *bfmR* (100%, n=96), followed by *bap* (99%, n=95), and *pgaA* (98%, n=94). *A. baumannii* strains were categorized into ten patterns based on the detected biofilm-related genes (Table 2). Sixty-nine (72%) strains harbored all the investigated genes and belonged to pattern 1. Additionally, at least seven biofilm-related genes were detected in all strains.

The relationship between the capacity of biofilm formation and the presence of biofilm-related genes is shown in Table 3. Our results underlined that the presence of *aba*I, *ade*B, *ade*J, *bap*, *bfm*S, *csu*E, and *pga*A genes had a significant relationship with biofilm production (P-values ranging from < 0.0001 to 0.014).

Molecular fingerprinting by ERIC-PCR. ERIC-PCR typing of the 96 *A. baumannii* isolates represented three distinct clonal types (CTs) and seven single types (STs), indicating a degree of genetic diversity among the strains. Among all, 38 strains (40%), 26 strains (27%), and 25 strains (26%) belonged to CT2, CT3, and CT1, respectively. Also, moderate biofilm producers showed more heterogeneity (6 types) than other strains (Table 4), and most biofilm-producing strains clustered in CT2.

DISCUSSION

Over the last decades, *A. baumannii* has been considered as a highly troubling cause of infection in burn patients worldwide due to its notable ability to achieve or upregulate numerous resistance determinants. In this work, the prevalence of XDR *A. baumannii* strains (99%) in burn wound infections was higher compared to those reported in the previous studies conducted in Iran (varying from 10-77%) (19, 20). The increased dissemination of XDR *A. baumannii* is attributed to the adaptive selection of drug-resistant isolates, a widespread diffuse of resistance determinants, cross-infection between inpatients, and misuse or excess use of broad-spectrum antimicrobials. Nevertheless, we found a lower level of resistance to amikacin, gentamicin, levofloxacin, and tetracycline compared to what had been seen before in Oman (21) and Iraq (22) but still higher than those reported in Mexico (23) and the United States (24). Differences between these findings are

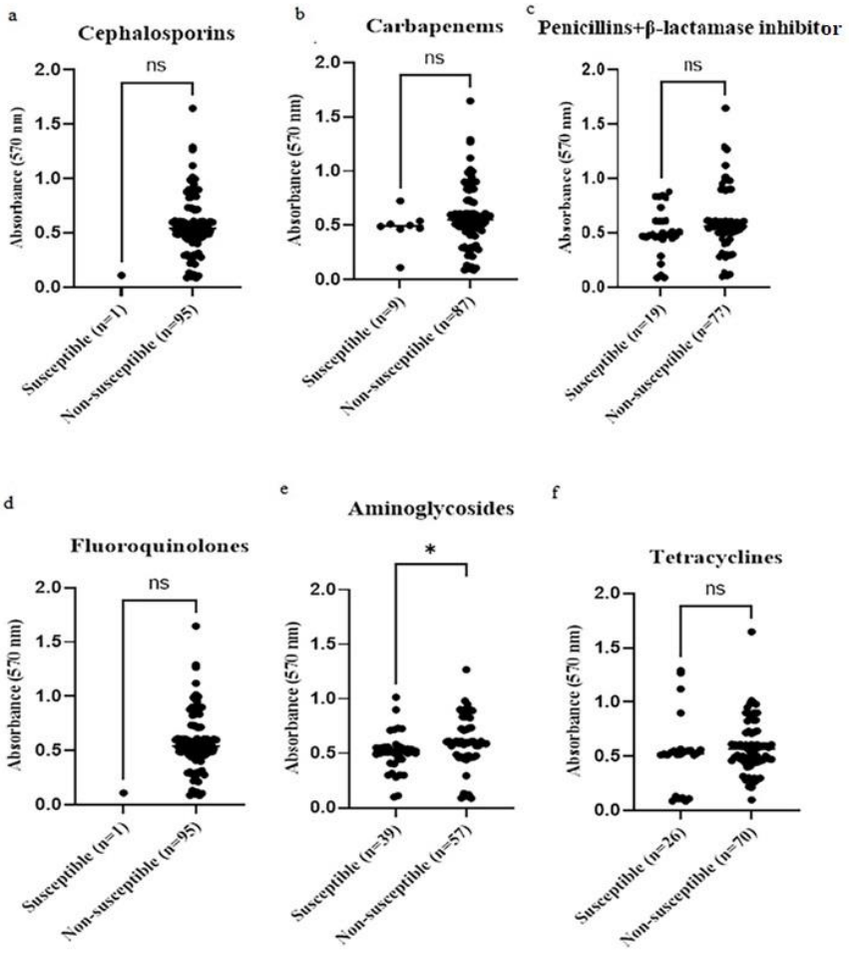

Fig. 2. Correlation between antibiotic susceptibility and production of biofilm across six antibiotic classes. Optical density at 570 nm (OD570, shows biofilm-forming ability. (a-f) a: cephalosporins, carbapenems, penicillins + β-lactamase inhibitors, fluoroquinolones, aminoglycosides, and tetracyclines. ns, *: Not significant and significant at $P \leq 0.05$.

Table 2. Prevalence of biofilm-related genes among the strains of *A. baumannii* categorized by biofilm-producing capacity.

Pattern	Genotype	Biofilm formation capacity				Total (%)
		Strong (%)	Intermediate (%)	Weak (%)	Non (%)	
1	*abaI, adeB, adeG, adeJ, bap, bfmR, bfmS, csuE, ompA, pgaA*	20 (83)	36 (67)	9 (82)	4 (57)	69 (72)
2	*abaI, adeB, adeJ, bap, bfmR, bfmS, csuE ompA pgaA*	-	6 (11)	2 (18)	2 (29)	10 (10)
3	*adeB, adeG, adeJ, bap, bfmR, bfmS, csuE, ompA pgaA*	-	4 (7)	-	-	4 (4)
4	*abaI, adeB, adeG, adeJ, bap, bfmR, bfmS, ompA pgaA*	2 (8)	-	-	-	2 (2)
5	*adeB, adeJ, bap, bfmR, bfmS, csuE ompA, pgaA*	2 (8)	-	-	-	2 (2)
6	*abaI, adeB, adeG, adeJ, bfmR, bfmS ompA, pgaA*	-	-	-	1 (14)	1 (1)
7	*adeB, adeG, adeJ, bap, bfmR, bfmS, csuE, ompA*	-	1 (2)	-	-	1 (1)
8	*abaI, adeJ, bfmR, bfmS, bap, csuE ompA, pgaA*	-	2 (4)	-	-	2 (2)
9	*abaI, adeB, adeG, bap, bfmR, csuE, ompA, pgaA*	-	2 (4)	-	-	2 (2)
10	*abaI, adeG, adeB, bap, ompA, bfmR, csuE*	-	3 (6)	-	-	3 (3)

Table 3. The association between biofilm formation and biofilm-related genes.

Biofilm-associated gene	Biofilm formation ability		P-Value
	Biofilm-producing No. (%)	Non-biofilm-producing No. (%)	
abaI			0.0068 (**)
Positive	81 (92)	7 (8)	
Negative	8 (100)	-	
adeB			0.014 (*)
Positive	87 (93)	7 (7)	
Negative	2 (100)	-	
adeG			0.097 (ns)
Positive	77 (94)	5 (6)	
Negative	12 (86)	2 (14)	
adeJ			0.0068 (**)
Positive	85 (92)	7 (8)	
Negative	4 (100)	-	
bap			<0.0001 (****)
Positive	89 (94)	6 (6)	
Negative	-	1 (100)	
bfmR			>0.9999 (ns)
Positive	89 (93)	7 (7)	
Negative	-	-	
bfmS			0.0068 (**)
Positive	85 (92)	7 (8)	
Negative	4 (100)	-	
csuE			<0.0001 (****)
Positive	87 (94)	6 (7)	
Negative	2 (67)	1 (33)	
ompA			>0.9999 (ns)
Positive	89 (93)	7 (7)	
Negative	-	-	
pgaA			0.0068 (**)
Positive	85 (92)	7 (8)	
Negative	4 (100)	-	

Significance cut-offs: ns, $P > 0.05$; *P, ≤ 0.05; **P ≤ 0.01, and ****, $P \leq 0.0001$.

often attributed to the differences in the surveillance programs, bacterial populations, or antibiotic consumption patterns among the countries. Current antibiotic therapy for burn wound infections in Iran is more based on empiric antibiotic regimens than antimicrobial susceptibility assays, and ciprofloxacin, ceftriaxone, imipenem, and piperacillin+tazobactam are among the most frequently prescribed drugs for these patients (25). According to our results, the most resistance was seen against imipenem, piperacillin/tazobactam, meropenem, ceftriaxone, ciprofloxacin, and cefotaxime which is consistent with previously published reports (11, 12, 19, 20, 25). Hence, our susceptibility data suggest that empirical therapy for wound infection in our country would be improper and there is an urgent need for modification and improvement in the drug prescription pattern based on area-specific antimicrobial resistance rates.

Biofilm formation is a great concern in burn-related infections, with biofilms contributing to approximately 60% of burn-associated mortality (26). In this context, our results revealed that over 92% of the tested *A. baumannii* strains formed biofilms, with 81% demonstrating strong or moderate biofilm-producing capacity. These findings are consistent with recent studies reporting biofilm formation rates in *A.*

Table 4. ERIC typing of the isolates as per the biofilm formation ability

ERIC-PCR pattern	Biofilm formation capacity			
	Non-biofilm former	Weak biofilm former	Intermediate biofilm former	Strong biofilm former
	No. (%)	No. (%)	No. (%)	No. (%)
CT1	1 (14)	5 (45)	16 (30)	3 (13)
CT2	-	-	20 (37)	18 (75)
CT3	5 (71)	4 (36)	15 (28)	2 (8)
ST1	-	1 (9)	-	-
ST2	1 (14)	-	-	-
ST3	-	-	-	1 (4)
ST4	-	-	1 (2)	-
ST5	-	-	1 (2)	-
ST6	-	-	1 (2)	-
ST7	-	1 (9)	-	-
Total	7	11	54	24

baumannii ranging from 75% to 100% (5, 27).

We observed a significant correlation between the production of biofilm and resistance to aminoglycosides. This could reflect the known role of aminoglycosides in inducing biofilm production in *A. baumannii* strains and adaptively responses to aminoglycoside exposure (28). For fluoroquinolones, carbapenems, tetracyclines, penicillins + β-lactamase inhibitors, and cephalosporins, significant differences in the production of biofilm between susceptible and non-susceptible strains were not seen. In line with our results, Hoffman et, al. showed that amikacin, tobramycin, gentamicin, and streptomycin induced biofilm production in *Pseudomonas aeruginosa*, while polymyxin B, chloramphenicol, and carbenicillin did not effect on biofilm production as inhibitors of the cell membrane, protein synthesis, and cell wall synthesis, respectively (29). Thus, biofilm induction by aminoglycosides is unlikely to be solely due to non-specific protein synthesis inhibition; rather, it appears to be a specific response by *A. baumannii* to aminoglycosides This finding aligns with previous research indicating that sub-inhibitory concentrations of aminoglycosides can induce biofilm production in *Escherichia coli*. Such a response is considered a specific bacterial defense mechanism against antibiotic stress and has been associated with changes in intracellular concentrations of cyclic di-GMP (c-di-GMP), a pivotal secondary messenger that controls surface attachment, biofilm maturation, and bacterial virulence (30). However, some studies have reported differing results, where exposure to certain aminoglycosides under specific conditions did not significantly enhance biofilm formation or even reduce it, depending on the bacterial species, antibiotic concentration, and environmental factors (31). These discrepancies highlight the complexity of antibiotic-biofilm interactions and suggest that the effects of aminoglycosides on the production of biofilm may be strain-dependent and influenced by various physiological and environmental variables (32).

Furthermore, analysis of the association between biofilm-related genes and the biofilm-forming capacity of *A. baumannii* strains revealed a highly significant correlation between the presence of *bap* and *csuE* genes and enhanced biofilm production ($p < 0.0001$). The pivotal roles of Bap, a biofilm-associated protein, and CsuE, the tip adhesin of the chaperone-usher pili system, in promoting biofilm development in *A. baumannii* have been extensively documented previously. Studies have demonstrated that deletion of *bap* significantly impairs biofilm maturation, leading to reduced biomass and loss of microcolony architecture (33). Similarly, mutants lacking *csuE* exhibit defective surface adhesion and initial biofilm formation (34). This supports the hypothesis that Bap and CsuE pili are essential not only for the initiation but also for the structural integrity of mature biofilms in clinical strains of *A. baumannii*. Our findings are consistent with previous reports and further reinforce the pivotal contribution of these factors to biofilm-associated persistence and pathogenicity. Therefore, these genes (*bap* and

csuE) may serve as promising therapeutic targets for managing biofilm-associated infections caused by *A. baumannii*, particularly in burn injuries. Additionally, understanding the local molecular epidemiology is essential for implementing effective infection control strategies. To the best of our knowledge, this work represents the first report of ERIC-PCR-based genotyping of strains of *A. baumannii* isolated from burn wound infections in Isfahan. The ERIC-PCR analysis represented a high level of genetic similarity among the strains, consistent with results from previous studies (35). The majority of the strains (89 of 96) were clustered in three ERIC types (CT1-CT3). It appears that these genotypes were the major circulating strains among burn-suffering patients in the hospital. These findings may have occurred as a result of cross-transmission of *A. baumannii* strains among patients via healthcare workers, equipment, or contaminated fomites (35). This study provides compelling evidence that the dual capacity of *A. baumannii* to develop antibiotic resistance and form robust biofilms enables this pathogen to persist in hospital environments, thereby promoting the spread of XDR strains within healthcare settings and potentially into the broader community. Because the majority of the strains were categorized as medium biofilm producers, these strains showed the most heterogeneity. Most biofilm-producing strains were classified within clonal type 2 (CT2), suggesting a possible association between the capacity of formation of biofilm and genetic clustering among *A. baumannii* isolates. While this research offers valuable insights, some limitations should be acknowledged. First, the investigation was geographically restricted to a single burn hospital in Isfahan, which can restrict the generalizability of the results to other healthcare settings. Also, to better comprehend the precise task of biofilm-associated genes in the production of biofilm and the development of antibiotic resistance, future studies should incorporate real-time PCR to determine gene expression levels under varying environmental conditions, particularly in the presence of antimicrobial agents.

CONCLUSION

This study represented the high prevalence of biofilm-producing XDR *A. baumannii* in burn wound infections, with a high frequency of biofilm-associated genes. Our results indicated that the *aba*I, *ade*B, *ade*J, *bap*, *bfm*S, *csu*E, and *pga*A genes influence biofilm formation. Also, we observed an association between the production of biofilm and resistance to aminoglycosides. During the study period, high genetic similarity was observed among *A. baumannii* strains, which can be considered as cross-transmission within patients. Given the huge social and economic burdens of infection in wound care after burn injury, comprehension of the definite relationship between drug resistance and the capacity to produce biofilm is vital for allotting resources for prevention and treatment.

ACKNOWLEDGEMENTS

The results presented in this paper were part of Sanaz Khashei's Ph.D. thesis funded by a research grant from the Office of Vice-Chancellor for Research and Technology, Isfahan University of Medical Sciences, Isfahan, Iran.

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