



## Detection of *qacE* and *qacEΔ1* genes in *Acinetobacter baumannii* and *Pseudomonas aeruginosa* isolated from hospital environments

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### Abstract

**Objective:** To determine the prevalence of *qacE* and *qacEΔ1* genes among *Acinetobacter baumannii* and *Pseudomonas aeruginosa* obtained from hospital environments, and assess their potential role in disinfectant resistance within healthcare settings. **Methods:** The bacterial isolates were obtained from different hospital surfaces and medical equipment, from medical city hospitals in Baghdad (Baghdad Teaching Hospital, Children Welfare Teaching Hospital, Ghazi Al-Hariri Hospital for surgical specialties, and teaching laboratories in Medical City). The disc diffusion method was used to determine the susceptibility of *A. baumannii* and *P. aeruginosa* strains to three different disinfectants. DNA was extracted from bacteria and conventional polymerase chain reaction (PCR) techniques were used for each DNA extracted sample in order to detect the presence of *qacE* and *qacEΔ1* and determine the prevalence of each gene among more resistant isolates. **Result:** The 24 *A. baumannii* and *P. aeruginosa* disinfectant-resistant isolates were molecularly analyzed using conventional PCR techniques. The *qacE* gene was present in 76.92% of *Acinetobacter baumannii* and 100% in *Pseudomonas aeruginosa* before disinfectant treatment, and decreased to 53.85% and 61.5% respectively after treatment. The *qacEΔ1* gene was detected in 15.3% of *A. baumannii* and 53.8% of *P. aeruginosa* isolates prior the treatment, but was completely absent in all isolates after disinfectant exposure. **Conclusion:** The study demonstrated that the prevalence of the *QacE* gene in *A. baumannii* and *P. aeruginosa* decreased after disinfectant treatment, while the *qacEΔ1* was completely lost. These findings emphasize the need to improve hospital disinfection strategies by optimizing disinfectant combinations and concentrations to target biofilm-forming bacteria. Routine monitoring of resistance genes such as *qacE* and *qacEΔ1* should be included in infection control programs. This suggests disinfectants can partially suppress resistance genes but fail to fully eliminate resistant strains, especially those with strong biofilm-forming ability.

**Keywords:** QACE, QACEΔ1, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*

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## Introduction

Healthcare-associated infections (HAIs) are defined as infections that arise during the course of healthcare delivery within a hospital or other healthcare facility, manifesting either 48 hours or more following a hospital admission or within a 30-day period subsequent to receiving healthcare services (Haque *et al.*, 2018). According to (Abreu *et al.*, 2013) the escalation of healthcare-associated infections is associated with an increased bacterial capacity to resist and adapt to adverse environmental conditions; this allows deadly pathogens to persist on hospital surfaces for extended periods, thereby transforming the environment into perpetual reservoir of infectious agents. Hospital common areas, including waiting rooms, restrooms, and elevators, are particularly critical due to the large number of people passing through them on a daily basis, despite the implementation of standard, methodical cleaning techniques, it is difficult to maintain the sanitization and safety of these areas, particularly door handles and surfaces (Cunningham *et al.*, 2022). In these environments, the most important approach is to prevent the transmission of pathogens, which is particularly important given the widespread distribution of multi-drug-resistant bacterial pathogens (Christaki, Marcou and Tofarides, 2020; Al-Jumaili and Ahmed, 2024).

The ESKAPE group, comprising *Klebsiella pneumoniae*, *Enterobacter* spp., *Enterococcus faecium*, *Staphylococcus aureus*, *Acinetobacter baumannii*, and *Pseudomonas aeruginosa* is a prominent etiological agent of severe

infections and constitute a major contributor to nosocomial infections, They can form biofilms on several abiotic surfaces that may account for their perseverance in the hospital environment, increasing the possibility of causing healthcare-associated infections and outbreaks (Ayoub Moubareck and Hammoudi Halat, 2020; Flores-Paredes *et al.*, 2021). These bacteria are mainly responsible for surgical site infection (SSI), bloodstream infection, pneumonia, odontogenic infection in the oral and maxillofacial regions, sepsis, and urinary tract infection (Michiels *et al.*, 2016; Park *et al.*, 2021; Ghaima *et al.*, 2018).

*Pseudomonas aeruginosa* is a Gram-negative, aerobic, motile bacterium with a slightly curved morphology and exhibits a ubiquitous presence across various environments, including water, air, soil, and both plant and animal tissues. (Chimi *et al.*, 2024). It stands as the fourth most frequently isolated nosocomial pathogen, contributing to 10% of all hospital-acquired infections, including pulmonary infections (tracheobronchitis and necrotizing bronchopneumonia), urinary tract infections, bacteremia, endocarditis, and skin and soft-tissue infections such as surgical and burns wounds (Caffrey *et al.*, 2022).

*Acinetobacter baumannii*, colloquially referred to as “Iraqibacter,” gained prominence during the Iraq War due to its association with infections in wounded U.S. military personnel, this gram-negative bacterium was frequently isolated from soft tissue infections in soldiers treated in field hospitals in Iraq and Afghanistan, this pathogen has demonstrated a remarkable ability to

persist in hospital environments, particularly in intensive care units (ICUs), where it poses a significant challenge due to its multidrug-resistant (MDR) nature (Peleg, Seifert and Paterson, 2008; AL-Masoudi, AL-Saffar and Kendla, 2015). The global dissemination of *A. baumannii* is attributed to its resilience and remarkable adaptability, thriving under diverse environments, including varying pH levels, and its ability to survive in dry environments and form biofilms (Ibrahim, 2019; Atta *et al.*, 2019). Initially regarded as a low-grade pathogen, it has evolved into a major cause of both nosocomial and community-acquired infections due to its ability to persist on medical devices such as surgical tools, ventilators, and catheters (Lucidi *et al.*, 2024; Moubareck and Halat, 2020).

The mechanisms underlying tolerance or resistance to disinfectants in bacteria involve several factors, including inherent structural characteristics of the bacterial cell wall, alteration in cell membrane structure and function, the activity of efflux pumps, the formation of biofilms, and the degradation of QACs (Boyce, 2023). The *qacE* and its variant *qacEΔ1* genes are widely distributed in gram-negative bacteria, especially those harboring class 1 integrons, which facilitate horizontal gene transfer (Chen *et al.*, 2023). The *qacE* gene encodes an efflux pump that confers resistance mainly to quaternary ammonium compounds and related disinfectants, but its presence does not always translate to high-level resistance alone; it often acts synergistically with other mechanisms, such as biofilm formation that protect

bacteria from disinfectants contributing to survival and persistence in clinical environments (Jaglic and Cervinkova, 2012).

Disinfectants and antiseptics play a major role in the control and prevention of these infections. Given the heavy reliance on disinfectants for hospital surface decontamination, the occurrence of *qacE* and *qacEΔ1* in *A. baumannii* and *P. aeruginosa* isolated from clinical environments warrants investigation. Detecting these genes provides insight into the potential inefficacy of commonly used antiseptics and highlights the risk of cross-resistance between disinfectants and antibiotics (Bessonneau *et al.*, 2013). Therefore, the study's aim specifically was to examine the distribution of *qacE* and *qacEΔ1* in these bacterial isolates obtained from hospital environments, offering critical information for infection prevention

## Materials and Methods

### *Bacterial Collection and Diagnosis*

Sixty bacterial samples were collected from various hospital surfaces and medical equipment, including neonatal incubators, surgical tools, patient beds, bed corners, urinary catheters, and air conditioning openings, and were isolated during the period from April 2023 to the end of March 2024. Thirteen isolates were *Acinetobacter baumannii*, and thirteen were *Pseudomonas aeruginosa*. All samples were obtained from medical city hospitals in Baghdad (Baghdad Teaching Hospital, Children Welfare Teaching Hospital, Ghazi Al-Hariri Hospital for surgical specialties, and teaching laboratories in Medical City). After collecting the samples, they were

immediately cultured in brain heart infusion broth to activate and permit the majority of the bacterial genus to grow. They were then incubated at 37 °C for 24 hours; a gram stain test was performed to differentiate between Gram-positive and Gram-negative bacteria and also cultured on MacConkey agar and blood agar plates to be considered selective media that allowed the growth of gram-negative bacteria as well as a differential medium for differentiating bacteria from fermented and nonfermented lactose. These plates were incubated overnight at 37 °C under aerobic conditions and examined the next day for bacterial

growth. The identification of bacterial isolates was confirmed using the modern colorimetric technology VITEK2-Compact, which includes 64 biochemical assays for bacterial identification

### 1.1 Disinfectants

Three different disinfectants were used in this experiment, as shown in Table 1, based on their documented use in sterilization protocols within the hospitals from which bacterial samples were sourced. *A. baumannii* and *P. aeruginosa* isolates were cultured to test their resistance to these disinfectants.

**Table1: Disinfectant formulations used in the study.**

Disinfectants	Active ingredient
First disinfectant	45% Ethyl Alcohol (Ethanol), 5% Propane -2-ol (Isopropyl Alcohol), Didecyl Dimethyl Ammonium Chloride (DDAC).
Second disinfectant	- step 1: aniosome used (Quaternary ammonium propionate, chlorhexidine digluconate, non-ionic surfactants, enzyme complex (protease, lipase and amylase). - step 2: steranios used (2% glutaraldehyde solution buffered at pH 6 (sodium citrate)
Third disinfectant	10% Didecyl Dimethyl Ammonium Chloride (DDAC), 2% benzalkonium chloride, anti-corrosive agent, and auxiliary substances

### 1.2 Detection of *qacE* and *qacEΔ1*

DNA was extracted using the Bacterial DNA Extraction kit (ELK Biotechnology, China) from thirteen *Acinetobacter baumannii* and thirteen *Pseudomonas aeruginosa*. The conventional PCR technique was carried out to amplify fragments of *qacE* and *qacEΔ1* using specific primer pairs (Table 2). 10 µl of master mix (Table 3), 5 µl of DNA sample, 1 µl (10 pmol/l) of

each forward and reverse primer, and 5 µl of free-nuclease water were added. The extracted DNA and primers were thawed at 4°C, vortexed, and then centrifuged to bring the contents to the tubes' bottoms. The reaction was carried out using the ideal PCR conditions for the genes indicated in the table (4). Agarose gel (2%) containing Ethidium Bromide was used to find out the PCR products, and a UV light was used to observe the gel.

**Table 2: The primers.**

N.O	Primer	Sequence (5'-3')	TM	Product size (bp)	Reference
1-	qacE	F: TTACTAAGCTTGCCCTTCCG R: TGAGCCCCATACCTACAAAGC	62	201	Designed in this study
2-	qacEΔ1	F: GCTGGCTTTTTCTTGTTATCGC R: GATCGGGCGAGCAAAAAGG	60	301	

**Table 3: Reaction setup and thermal cycling protocol.**

N. O	Master mix components	Volume (μl)
1-	Enti Link™ PCR Master Mix (Blue)	10
2-	Forward primer	1
3-	Reverse primer	1
4-	DNA	3
5-	Nuclease-free water	5
	Total volume	20 μl

**Table 4: The ideal PCR condition.**

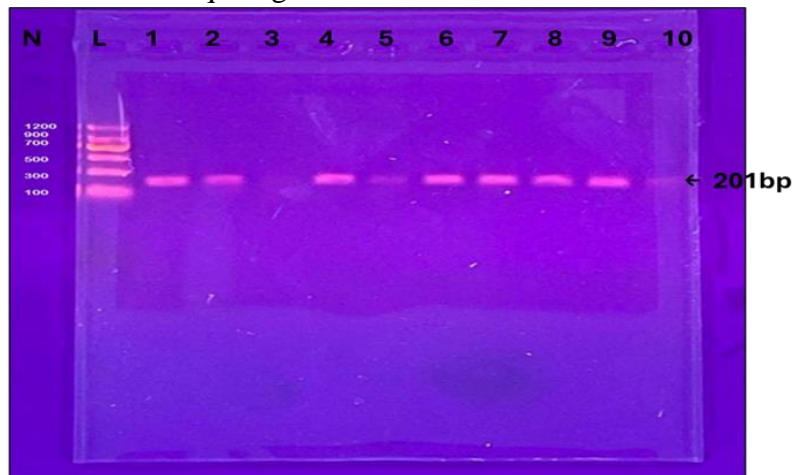
Steps	Temperature	Time (minutes: seconds)	Cycle No.
Initial denaturation	94	05:00	1
Denaturation	94	00:20	45x
Annealing qacE	62	00:20	
Annealing qacEΔ1	60	00:20	
Extension	72	00:20	
Final extension	72	10:00	1

## Results

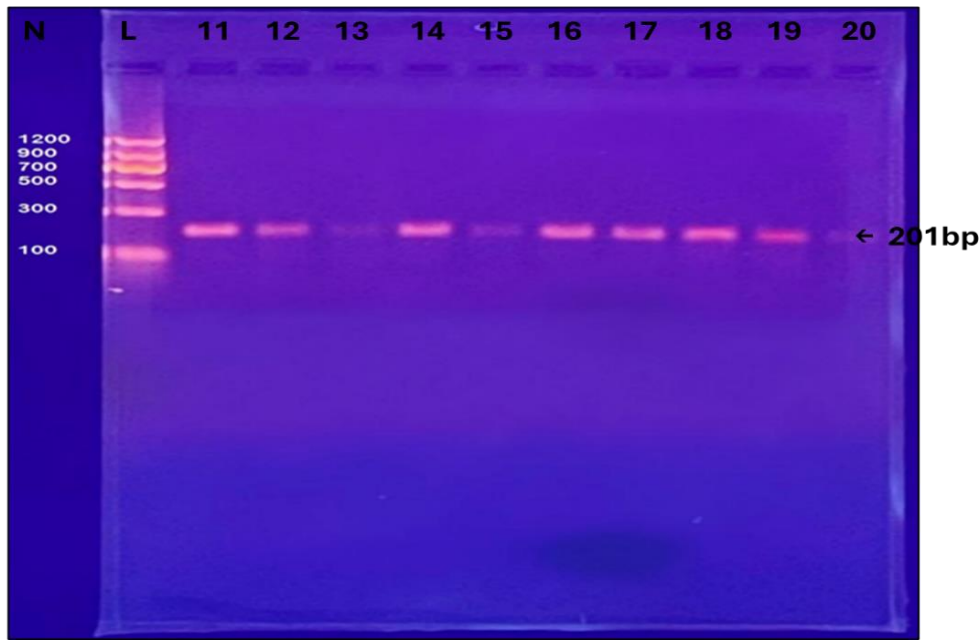
### 1.1 QacE

The isolates of all the detected resistant bacteria were molecularly analyzed using conventional PCR techniques. The results found ten out thirteen isolates of *A. baumannii* contained the qacE gene before the treatment (76.92%), seven of them seemed to have the qacE gene after

the treatment (53.85%), in *P. aeruginosa* all the 13 isolates have the qacE gene before the treatment, but 8 of them have the gene after the treatment (61.5%), obvious band on agarose gel was appeared and expected size as 201 bp in the distance between 200 and 300bp of DNA ladder bands as it shown in figure 1(a),1(b)



**Figure 1(a): Gel electrophoresis of PCR product to qacE gene. 2% agarose gel at 50 volt for 40 minutes, then visualized under UV after staining with ethidium bromide.**

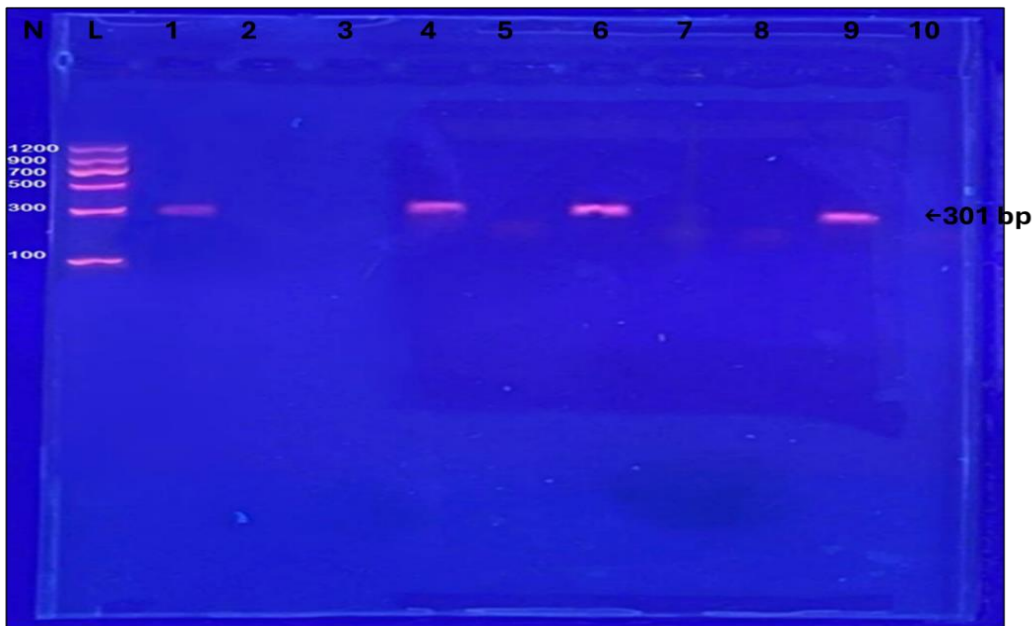


**Figure 1(b):** Gel electrophoresis of the PCR product for the *qacE* gene. A 2% agarose gel was run at 50 volts for 40 minutes, then visualized under UV light after staining with ethidium bromide.

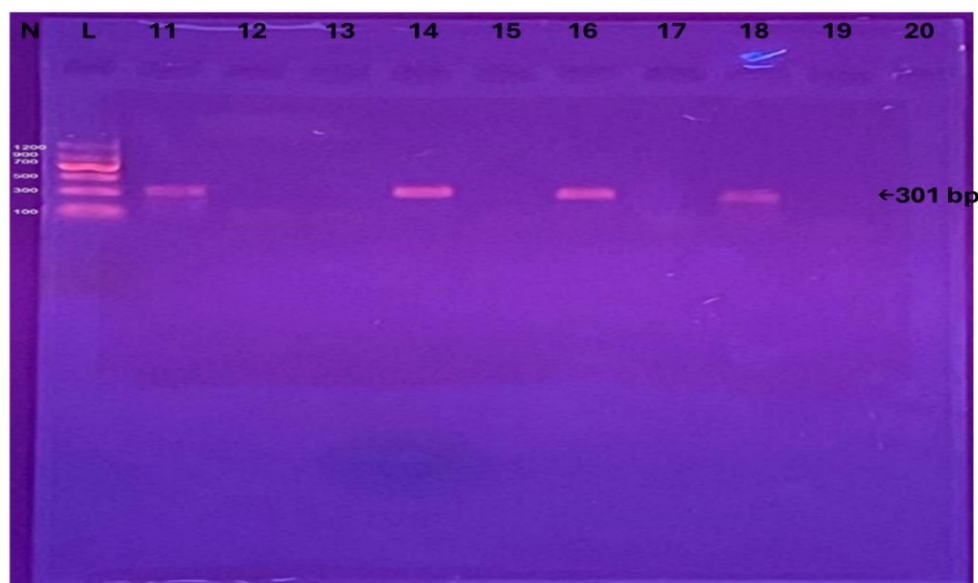
### 1.2 *QacEA1*

One of the objectives of the present study is to detect the existence and prevalence of *QacEA1*. Before the treatment with disinfectants, the gene was detected in 5 out of 13 isolates (15.3%) of *A. baumannii* strain, clear band on agarose

gel was appeared and expected size as 301 bp above 300 bp of the DNA ladder bands. In *P. aeruginosa*, 8 out of 13 isolates had the *qacΔE1* gene before the treatment (53.8%). No existence of *QacEA1* gene was detected in any of the isolates after disinfectant application as it shown in figure 2(a),2(b).



**Figure 2(a):** Gel electrophoresis of the PCR product for the *qacEA1* gene. A 2% agarose gel was run at 50 volts for 40 minutes, then visualised under UV light after staining with ethidium bromide. 301 base pair.



**Figure 2(b):** Gel electrophoresis of the PCR product for the *qacEΔ1* gene. A 2% agarose gel was run at 50 volts for 40 minutes, then visualised under UV light after staining with ethidium bromide. 301 base pair.

The molecular detection results of the *qacE* and *qacEΔ1* genes among *Acinetobacter baumannii* and *Pseudomonas aeruginosa* isolates both prior and post disinfectant exposure.

Table 5 show a partial decrease in the prevalence of *qacE* and complete loss of *qacEΔ1* post disinfectant exposure, suggesting an application of selective pressure by disinfectant exposure.

**Table 5: Prevalence of QACE and QACEΔ1 genes in a. baumannii and p. aeruginosa before and after disinfectant treatment.**

Bacterial Species	Gene	Before Treatment (n = 13)	% Positive Isolates	After Treatment (n = 13)	% Positive Isolates	Change After Treatment
<i>A. baumannii</i>	<i>qacE</i>	10/13	76.92%	7/13	53.85%	↓ 23.07%
<i>A. baumannii</i>	<i>qacEΔ1</i>	2/13	15.30%	0 / 13	0%	Complete loss
<i>P. aeruginosa</i>	<i>qacE</i>	13 / 13	100%	8/13	61.50%	↓ 38.5%
<i>P. aeruginosa</i>	<i>qacEΔ1</i>	7/13	53.80%	0 / 13	0%	Complete loss

## Discussion

This research aimed to expand the scope of examination to detect the resistance mechanisms of these bacteria in healthcare environments against disinfectants after the previous findings of the effect of the local disinfectants on the ability of these bacteria to form biofilm the result showed that most of *Acinetobacter baumannii* and

*Pseudomonas aeruginosa* isolates demonstrated strong and moderate biofilm across all disinfectant interventions, with a few instances of weak biofilm formation observed in the second disinfectant application (Radi Atta, Al Haideri and H Murbat, 2019; Abbas and Mohammed, 2025). The observed decline in the *QacE* gene in most of the strains after disinfectant use suggests either partial gene loss or

suppression of gene expression, a dynamic that varies with disinfectant type, concentration, and exposure time. The difference in *qacE* gene prevalence between species may also reflect differing intrinsic resistance procedures, plasmid carriage rates, or environmental exposure histories.

Furthermore, strong and moderate biofilm formation seemed to be an important trait in most isolates in terms of reducing effectiveness of disinfectants since causing a physical and biochemical barrier to penetration. This protection from biofilm can increase the likelihood of cross-resistance in disinfectants and antibiotics, as efflux pumps and stress response systems may be co-regulated under biofilm conditions.

The findings are in agreement with (Guo *et al.*, 2015), who found that 55.5% *K. pneumoniae* persistence linked this gene to carbapenem-resistant strains. The post-treatment result of *qacE* prevalence suggests that disinfectants containing QACs and glutaraldehyde may selectively pressure susceptible strains, thereby only selecting for the resistant populations, especially in biofilm-forming bacteria like *P. aeruginosa* and *A. baumannii*, which are known for their resilience (Kazama *et al.*, 1998; Chen *et al.*, 2023)

The results are in agreement with (Kosyakova *et al.*, 2020), who found varying prevalence of the *qacE* gene among resistant bacteria. *A. baumannii* showed a decrease from 76.92% to 53.85% in *qacE* presence after treatment, which suggested a complex relationship between disinfectant exposure and resistance gene retention, with biofilm formation being moderate to strong,

which improves bacterial survival against disinfectants. The findings align with those of (Gülbudak *et al.*, 2023). Who noted that out of 44 *P. aeruginosa* carbapenem-resistant isolates, 75.8% of them had the *qacE* gene.

The complete loss of *qacE* $\Delta$ 1 after disinfectant application contrasts with (Wang *et al.*, 2025) study, which showed that sublethal exposure to disinfectants often selects for resistance genes. In the current study, despite gene loss, biofilm robustness underscores the insufficient disinfection process, as extracellular polymeric substance (EPS) and metabolic adaptations maintain bacterial survival.

These results have significant implications for infection control in healthcare settings. The ability of *qacE*-positive cultures to survive disinfectant challenges means that traditional cleaning regimens may not be able to remove resistant bacteria from hospital surfaces. As a result, there is a clear need for institutions to regularly monitor the efficacy of their disinfectant selection, rotate active compounds to avoid adaptive responses, and rigorously enforce various cleaning and disinfecting protocols in high-risk areas, especially in intensive care units. Further, the use of molecular surveillance to monitor for resistance genes may help to rapidly detect and control the spread of disinfectant-tolerant resistant strains.

The outcomes disagree with (Al-Azzawi, 2018), who isolated 67 *Pseudomonas aeruginosa* from wounds and burn patients treated with quaternary ammonium compound disinfectant and found that 97.1% of the isolates had *QacE* and *QacE* $\Delta$ 1 genes.

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*Authors' Contribution:* Both authors conceived and designed the study. Abbas performed experiments, analyzed the data, and wrote the manuscript. Mohammed was involved in data analysis and manuscript correction. Both authors have read and approved the final manuscript.

*Data Availability:* The data used in this study (environmental bacterial samples) were collected from the hospitals by the researchers after obtaining the required official approvals from the hospital administrations.

*Study Registration:* This study was not registered in an external registry, as it does not involve clinical trials or interventional studies requiring prior registration. All procedures were conducted after receiving official approval from the hospitals.

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