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RESEARCH ARTICLE OPEN ACCESS

Antibiotic resistance patterns and associated genetic determinants among clinical isolates of Pseudomonas aeruginosa from a tertiary hospital in southern Iraq

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Abstract
Objective: To evaluate antibiotic resistance patterns and investigate the distribution of the carbapenemase-associated gene *blaOXA-23* and the biofilm-associated gene *algD* among clinical isolates of *Pseudomonas aeruginosa* recovered from patients at a tertiary hospital in Southern Iraq.
Methods: A cross-sectional study was conducted from November 2025 to April 2026 at Al-Nasiriyah Teaching Hospital. A total of 120 *P. aeruginosa* isolates were collected from clinical specimens (burned tissue, sputum, urine, bronchoalveolar lavage, and others). Antimicrobial susceptibility was tested against 14 agents using the VITEK 2 system. Isolates were categorised as non-MDR, MDR, XDR, and pandrug-resistant (PDR). PCR was used to detect *blaOXA-23* and *algD*. Statistical analysis included chi-square tests, with Cramér's V for effect sizes.
Results: PDR isolates were most prevalent (33.3%), followed by XDR (24.2%), non-MDR (23.3%), and MDR (19.2%). Burned tissue yielded the highest proportion of PDR (n=18). Resistance rates were alarmingly high for ceftazidime (78.3%), cefotaxime (73.3%), and carbapenems (≈50%). *algD* alone was detected in 42.5% of isolates, *blaOXA-23* alone in 15.8%, and co-occurrence in 22.5%. Significant associations were found between resistance patterns and specimen type ($p<0.01$, Cramér's $V=0.31$), hospitalisation status ($p<0.01$, $V=0.36$), and age group ($p<0.05$, $V=0.24$).
Conclusions: The high prevalence of MDR, XDR and PDR *P. aeruginosa* in clinical samples underscores the urgent need for integrated surveillance. The co-occurrence of *blaOXA-23* and *algD* in highly resistant isolates suggests a synergistic interplay between carbapenemase activity and biofilm-mediated protection.

Keywords: *Pseudomonas aeruginosa,* antimicrobial resistance, pandrug resistance, biofilm, OXA-23, Iraq

Plain English Summary

Pseudomonas aeruginosa is a bacterium that often causes severe infections in hospitalised patients, especially those with burns or on ventilators. It is difficult to treat because it can resist many antibiotics. This study looked at 120 samples from patients in a teaching hospital in southern Iraq. We tested which antibiotics the bacteria could resist and looked for two specific genes: one that helps the bacteria destroy a last-line antibiotic (carbapenems), and another that helps the bacteria form a protective layer called biofilm. We found that one-third of the isolates were resistant to all antibiotics tested – a situation called pandrug resistance. Burn patients were most affected. The gene for biofilm formation was very common, and when both genes were present together, the bacteria were almost always highly resistant. This study shows that Iraqi hospitals need better infection control and regular monitoring of antibiotic resistance to prevent these dangerous bacteria from spreading further.

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Introduction

Antimicrobial resistance (AMR) is one of the greatest threats to global health in the 21st century. Hospital environments act as reservoirs for multidrug-resistant (MDR) microorganisms, and *Pseudomonas aeruginosa* stands out as a major opportunistic pathogen responsible for hospital-acquired infections (HAIs) worldwide (1, 2). This organism possesses both intrinsic and acquired resistance mechanisms, making it notoriously difficult to eradicate and leading to high morbidity and mortality among vulnerable patients (3).

The clinical challenge posed by *P. aeruginosa* is compounded by its ability to form biofilms – structured communities of bacteria encased in a self-produced matrix. Biofilms not only protect bacteria from host immune responses but also dramatically reduce the effectiveness of antibiotics (4). A key gene involved in biofilm formation is *algD*, which encodes a critical enzyme for alginate production. Overexpression of *algD* promotes the mucoid phenotype and is strongly associated with enhanced antimicrobial tolerance (5).

In addition to biofilm-mediated protection, enzymatic resistance mechanisms, particularly carbapenemase production, have become increasingly important. Carbapenems are considered last-line antibiotics for severe Gram-negative infections. Among the various carbapenemases, OXA-type enzymes – including OXA-23 – have emerged as clinically significant determinants of carbapenem resistance. Although *blaOXA-23* is classically associated with *Acinetobacter baumannii*, several recent reports have documented its presence in *P. aeruginosa* isolates from the Middle East, including Iraq (6, 7). This cross-species dissemination raises serious concerns about horizontal gene transfer in hospital settings.

The World Health Organisation has listed carbapenem-resistant *P. aeruginosa* as a critical priority for new antibiotic development (8). However, in low- and middle-income countries, surveillance data combining both phenotypic and molecular analyses remain scarce. Iraq, with its history of conflict and strained healthcare infrastructure, faces particular challenges in combating AMR (9, 10). The present study was therefore designed to assess antibiotic resistance patterns in *P. aeruginosa* isolated from clinical specimens at a tertiary hospital in southern Iraq and to explore the distribution of *blaOXA-23* and *algD* in relation to resistance phenotypes and patient characteristics.

Materials and Methods

Study design and setting

This was a cross-sectional descriptive study conducted at Al-Nasiriyah Teaching Hospital, Thi-Qar Health Directorate, Thi-Qar Province, southern Iraq. The hospital is a major referral centre serving both urban and rural populations. The study period extended from 15 November 2025 to 1 April 2026.

Sample collection and study population

A total of 120 non-duplicate clinical isolates of *P. aeruginosa* were collected from 120 patients attending the hospital. The sample comprised 86 inpatients and 34 outpatients. Inclusion criteria were any patient from whom a clinical sample yielded *P. aeruginosa* during the study period, regardless of age, sex or ward. Specimens were categorised into five types: burned tissue or wound swabs (n=39), sputum (n=26), mid-stream urine (n=19), bronchoalveolar lavage fluid (n=17), and other specimens (blood cultures, non-burn wound discharges, ear swabs; n=19). Demographic and clinical data – including age, sex, hospitalisation status, and ward – were recorded systematically.

Bacteriological culture and isolation

All specimens were transported to the laboratory within 2 hours of collection. Samples were inoculated onto blood agar, cetrimide agar, MacConkey agar, and brain-heart infusion broth. Cultures were incubated aerobically at 37°C for 24–48 hours. Colonies suspicious for *P. aeruginosa* were identified by Gram staining, oxidase test, pyocyanin production, and typical colonial morphology on cetrimide agar. All isolates were confirmed using the API 20NE system (bioMérieux, France). For quality assurance, *P. aeruginosa* ATCC 27853 was used as a reference strain.

Automated identification and antimicrobial susceptibility testing

Species confirmation and antimicrobial susceptibility testing were performed using the VITEK 2 Compact system (bioMérieux, France). Fresh overnight colonies were suspended in 0.45% sterile saline to a turbidity of 0.50–0.63 McFarland. Identification was carried out with the GN identification card. Susceptibility testing used the AST-N419 card, which contains 14 clinically relevant antimicrobial agents: piperacillin/tazobactam, ceftazidime, ceftazidime/avibactam, ceftolozane/tazobactam, cefepime, cefotaxime, imipenem, meropenem, amikacin, gentamicin, netilmicin, ciprofloxacin, aztreonam, and piperacillin. Results (sensitive, intermediate, or resistant) were interpreted according to Clinical and Laboratory Standards

Institute (CLSI) guidelines (M100-S34, 2024 edition). Quality control was performed using *P. aeruginosa* ATCC 27853.

Based on the number of antibiotic classes to which each isolate was resistant, we classified the isolates into four phenotypes following the definitions proposed by Magiorakos et al. (7): non-MDR (susceptible or resistant to ≤2 classes), MDR (resistant to ≥3 but not all classes), XDR (resistant to all but ≤2 classes), and PDR (pandrug-resistant; resistant to all tested classes). Intermediate results were considered resistant for classification purposes.

Molecular detection of blaOXA-23 and algD

Genomic DNA was extracted from pure cultures using the Genomic DNA Mini Kit (Geneaid, Taiwan) according to the manufacturer's instructions. Conventional PCR was performed to detect the carbapenemase gene *blaOXA-23* and the biofilm-associated gene *algD*. Primer sequences and expected product sizes are shown in Table 1. PCR reactions included positive controls (genomic DNA from known *blaOXA-23*-positive *A. baumannii* and *algD*-positive *P. aeruginosa*) and negative controls (sterile water). Thermal cycling conditions for *blaOXA-23* were: initial denaturation at 94°C for 3 min; 35 cycles of 95°C for 25 s, 52°C for 45 s, and 72°C for 50 s; final extension at 72°C for 5 min. For *algD*: initial denaturation at 95°C for 2 min; 35 cycles of 95°C for 1 min, 55°C for 33 s, 72°C for 1 min; final extension at 72°C for 5 min. Amplicons were resolved on 1.5% agarose gels stained with ethidium bromide and visualised under UV light. PCR product sizes were confirmed by comparison with a 100-bp DNA ladder. No sequencing was performed; therefore, the presence of *blaOXA-23* is reported based on amplicon size only.

Table 1: Primer sequences used for PCR detection

Gene	Primer	Sequence (5′→3′)	Product size	Reference
blaOXA-23	F	TGGAAGGGCGAGAAAAGGTC	~400 bp	(11)
blaOXA-23	R	TTGCCCAACCAGTCTTTCCA		
algD	F	TTCCCTCGCAGAGAAAACATC	~500 bp	(12)
algD	R	CCTGGTTGATCAGGTCGATCT		

Statistical analysis

Data were analysed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Categorical variables were compared using the chi-square (χ^2) test. For significant associations, effect sizes were calculated using Cramér's V (small ≥0.1, medium ≥0.3, large ≥0.5). Odds ratios with 95% confidence intervals were computed for selected binary comparisons (e.g., PDR vs. non-PDR by hospitalisation status). No adjustment for multiple comparisons was made because the analyses were exploratory; however, this limitation is acknowledged in the discussion. A p-value ≤0.05 was considered statistically significant.

Results

Characteristics of the study population

Among the 120 patients, 86 (71.7%) were inpatients, and 34 (28.3%) were outpatients. The mean age was 42.3 years (range 2–85 years). Age distribution was as follows: ≤20 years (n=32, 26.7%), 21–40 years (n=38, 31.7%), 41–60 years (n=28, 23.3%), and ≥61 years (n=22, 18.3%). Sex distribution was 67 males (55.8%) and 53 females (44.2%). The most common specimen types were burned tissue (32.5%) and sputum (21.7%).

Resistance phenotype distribution

Of the 120 isolates, pandrug-resistant (PDR) strains were the most prevalent (n=40, 33.3%), followed by XDR (n=29, 24.2%), non-MDR (n=28, 23.3%), and MDR (n=23, 19.2%) (Figure 1). The proportion of PDR isolates among inpatients (43.0%) was substantially higher than among outpatients (8.8%).

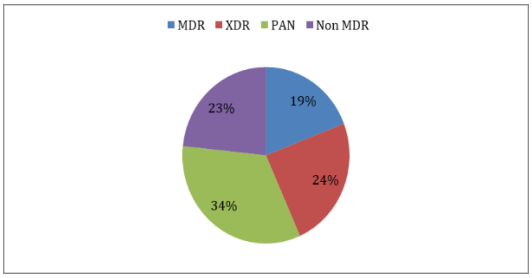

Figure 1: Distribution of antimicrobial resistance phenotypes (non-MDR, MDR, XDR, PDR) among 120 *P. aeruginosa* isolates.

Association with specimen type and patient status

The distribution of resistance phenotypes varied significantly by specimen type (χ^2=26.8, p<0.01, Cramér's V=0.31). Burned tissue specimens contributed the highest number of PDR isolates (n=18), followed by sputum (n=10) and bronchoalveolar lavage (n=6). In contrast, urine specimens showed a predominance of XDR isolates (n=10) (Table 2). Hospitalisation status was strongly associated with resistance: inpatients were far more likely to harbour XDR or PDR isolates than outpatients (χ^2=28.63, p<0.01, Cramér's V=0.36). The odds of being PDR among inpatients compared with outpatients was 7.8 (95% CI: 2.2–27.4). Ward-specific analysis (burn unit, ICU, RCU, medical wards) showed no statistically significant difference (p>0.05), although the burn unit accounted for the largest absolute number of PDR isolates (n=17).

Table 2: Distribution of resistance phenotypes by specimen type

Specimen type	Non-MDR	MDR	XDR	PDR	Total
Burned tissue	10	1	10	18	39
Sputum	7	5	4	10	26
Urine	4	10	4	1	19
BAL	2	4	5	6	17
Others	5	3	6	5	19
Total	28	23	29	40	120

χ^2=26.8, p<0.01, Cramér's V=0.31

Antibiotic susceptibility profile

Resistance rates were alarmingly high across the 14 antimicrobial agents tested (Table 3). The highest resistance was observed for ceftazidime (78.3%), cefotaxime (73.3%), ceftazidime/avibactam (69.2%), ciprofloxacin (66.7%), and cefepime (66.7%). Carbapenem resistance, a marker of last-line treatment failure, was found in 49.2% of isolates for imipenem and 50.0% for meropenem. Aminoglycoside resistance ranged from 60.8% to 63.3%. The overall variation in resistance rates across antibiotics was highly significant (χ^2=65.76, p<0.01).

Table 3: Antibiotic susceptibility profile of 120 *P. aeruginosa* isolates

Antimicrobial agent	Susceptible (n)	Intermediate (n)	Resistant n (%)
Piperacillin/tazobactam	42	10	68 (56.7)
Ceftazidime	23	3	94 (78.3)
Ceftazidime/avibactam	32	5	83 (69.2)
Ceftolozane/tazobactam	38	1	81 (67.5)
Cefepime	38	2	80 (66.7)
Cefotaxime	28	4	88 (73.3)
Imipenem	59	2	59 (49.2)
Meropenem	57	3	60 (50.0)
Amikacin	42	3	75 (62.5)
Gentamicin	42	5	73 (60.8)
Netilmicin	42	2	76 (63.3)
Ciprofloxacin	39	1	80 (66.7)
Aztreonam	43	6	71 (59.2)
Piperacillin	41	3	76 (63.3)

Molecular detection of blaOXA-23 and algD

Overall, 97 isolates (80.8%) carried at least one of the two target genes. The biofilm-associated gene *algD* was the most frequently detected determinant, present alone in 51 isolates (42.5%). The carbapenemase gene *blaOXA-23* was detected alone in 19 isolates (15.8%). Co-occurrence of both genes was found in 27 isolates (22.5%). No target gene was detected in 23 isolates (19.2%) (Table 4). Representative gel images are shown in Figures 2 and 3 (supplementary data).

Table 4: Distribution of target genes among 120 isolates

Gene profile	n	%
algD only	51	42.5
blaOXA-23 only	19	15.8
blaOXA-23 + algD	27	22.5
None detected	23	19.2

Association between gene profiles and resistance phenotypes

The distribution of gene profiles differed significantly among resistance categories (χ^2=23.56, p<0.01, Cramér's V=0.28). PDR isolates showed the highest frequency of co-occurrence (n=13, 32.5% of PDR) and the highest frequency of *algD* alone (n=19, 47.5%). XDR isolates also had notable co-occurrence (n=11, 37.9%). In contrast, non-MDR isolates most often lacked both genes (n=10, 35.7%) or carried only *algD* (n=13, 46.4%) (Table 5). These findings suggest a stepwise accumulation of resistance determinants as phenotypes progress from non-MDR to PDR.

Table 5: Gene profiles stratified by resistance phenotype

Phenotype	algD+blaOXA-23	blaOXA-23 only	algD only	None	Total
Non-MDR	2	3	13	10	28
MDR	1	5	10	7	23
XDR	11	5	9	4	29
PDR	13	6	19	2	40
Total	27	19	51	23	120

χ^2=23.56, p<0.01, Cramér's V=0.28

Age and sex distribution of resistance

Age group analysis (based on available data) showed a statistically significant association (χ^2=18.79, p<0.05, Cramér's V=0.24). PDR isolates were most frequent in the youngest age group (≤20 years, n=15) and the 21–40 years group (n=12). In contrast, MDR isolates were most common among patients aged ≥61 years (n=12). No significant difference in resistance phenotype distribution was observed between male and female patients (χ^2=1.87, p>0.05).

Discussion

In this study of 120 clinical *P. aeruginosa* isolates from a tertiary hospital in southern Iraq, we observed an extraordinarily high prevalence of pandrug-resistant (PDR) strains (33.3%) and extensively drug-resistant (XDR) strains (24.2%). These figures are among the highest reported from the Middle East and far exceed global pooled estimates, which typically range from 5% to 15% for PDR (13, 14). The predominance of PDR isolates from burned tissue (18 out of 40 PDR isolates) is particularly concerning. Burn patients are immunocompromised and frequently receive prolonged broad-spectrum antibiotic therapy, creating an intense selective pressure that favours the emergence and spread of pan-resistant clones (15, 16).

The strong association between inpatient status and PDR/XDR phenotypes (odds ratio 7.8 for PDR among inpatients) reinforces the role of hospitals as amplifiers of resistance. The burn unit, intensive care unit and respiratory care unit were hotspots for highly resistant isolates, although differences between wards did not reach statistical significance, possibly because of limited sample size per ward. Previous studies from Iraq have similarly identified burn units as epicentres of carbapenem-resistant *P. aeruginosa* (10, 17, 18, 19).

Our antibiotic susceptibility data reveal a disquieting landscape. Resistance rates to ceftazidime (78.3%) and cefotaxime (73.3%) are among the highest in the region. Even more alarming is the ~50% carbapenem resistance, as these drugs are often the last effective agents against multidrug-resistant Gram-negative infections (20). Such high rates severely limit empirical treatment options and compel clinicians to rely on older, more toxic agents such as colistin, which was not tested in this study but is likely the only remaining option for many PDR isolates.

Molecular findings: blaOXA-23 and algD

The detection of *blaOXA-23* in 38.3% of isolates (alone or in combination) requires careful interpretation. While *blaOXA-23* is classically considered an *Acinetobacter baumannii*-associated carbapenemase, several recent investigations from Iraq and neighbouring countries have reported its presence in *P. aeruginosa* clinical isolates (6, 7, 11). Jasim et al. (2024) identified *blaOXA-23* in *P. aeruginosa* from both cows and humans in Basra province (6). The most plausible explanation is horizontal gene transfer via plasmids or transposons that circulate among Gram-negative pathogens in hospitals with high antibiotic pressure. However, because we did not perform sequencing or conjugation experiments, we cannot definitively rule out non-specific PCR amplification. Nevertheless, the strong association between *blaOXA-23* positivity and carbapenem resistance (seen in Table 3 and Table 5) supports its functional role.

More than two-thirds of isolates (67.5%) carried the biofilm-associated gene *algD*, either alone or together with *blaOXA-23*. It is important to emphasise that *algD* is not an antimicrobial resistance gene per se; rather, it contributes to

the mucoid phenotype and biofilm formation, which physically protect bacteria from antibiotic penetration and promote tolerance (5, 21, 22). The presence of *algD* was not exclusive to resistant isolates; it was also found in 46.4% of non-MDR isolates, but its prevalence increased markedly in XDR and PDR groups. This pattern suggests that biofilm formation may act as a 'platform' that facilitates the survival and subsequent acquisition of bona fide resistance genes (4, 23).

The co-occurrence of *blaOXA-23* and *algD* in 13 PDR isolates (32.5% of PDR) and 11 XDR isolates (37.9% of XDR) is striking. While we cannot infer causality, the synergy between enzymatic carbapenem destruction and biofilm-mediated physical protection is biologically plausible: the biofilm matrix restricts antibiotic diffusion, reducing the effective concentration reaching bacterial cells, while any surviving cells that produce OXA-23 can hydrolyse carbapenems that do penetrate. This combination creates a highly refractory infection phenotype, as previously described in cystic fibrosis lung infections (5).

Age-related findings

The observation that PDR isolates were most common among patients ≤40 years of age, whereas MDR predominated in the elderly (≥61 years), is intriguing. A plausible explanation is that younger patients in this setting were more likely to be burn victims (burn injuries occur predominantly in children and young adults), and burn units have the highest antibiotic selection pressure. Conversely, older patients often present with chronic conditions and may have had multiple prior antibiotic exposures that select for MDR but not yet PDR strains. Unfortunately, our dataset did not allow us to stratify by both age and specimen type simultaneously, so this hypothesis remains speculative.

Study limitations and future directions

Several limitations must be acknowledged. First, this was a single-centre study, limiting generalisability to other hospitals in Iraq or the region. Second, the sample size (120 isolates) was moderate, and no a priori power calculation was performed. Third, we examined only two genetic determinants; many other resistance mechanisms (e.g., efflux pumps, porin loss, other β-lactamases) were not investigated. Fourth, we did not perform minimum inhibitory concentration (MIC) determinations – we relied on VITEK 2 categorical results. Fifth, PCR products were not sequenced; thus, the possibility of non-specific amplification of *blaOXA-23* cannot be entirely excluded. Sixth, we did not assess biofilm formation directly (e.g., by crystal violet assay), so the functional role of *algD* carriage remains inferred.

Future studies should include whole-genome sequencing to confirm resistance gene identities and explore the genetic context (plasmids, integrons). Multicentre surveillance with standardised protocols is urgently needed to map the true burden of PDR *P. aeruginosa* in Iraq. Additionally, clinical outcome studies should examine whether patients infected with *blaOXA-23*-positive isolates have worse outcomes than those with *blaOXA-23*-negative strains.

Conclusions

This study demonstrates that pandrug-resistant and extensively drug-resistant *P. aeruginosa* are alarmingly prevalent among hospitalised patients in southern Iraq, particularly those with burn injuries. The biofilm-associated gene *algD* is nearly ubiquitous, and its co-occurrence with the carbapenemase gene *blaOXA-23* is strongly associated with the most resistant phenotypes. While *algD* itself is not a resistance gene, its role in biofilm formation likely creates a microenvironment that favours the survival and persistence of carbapenemase-producing strains. These findings underscore the urgent need for integrated molecular surveillance, stricter infection control measures, and antibiotic stewardship programmes in Iraqi hospitals.

List of Abbreviations

AMR: Antimicrobial resistance
BAL: Bronchoalveolar lavage
CI: Confidence interval
CLSI: Clinical and Laboratory Standards Institute
HAIs: Hospital-acquired infections
ICU: Intensive care unit
MDR: Multidrug-resistant
MIC: Minimum inhibitory concentration
OR: Odds ratio
PCR: Polymerase chain reaction
PDR: Pandrug-resistant
RCU: Respiratory care unit
XDR: Extensively drug resistant

Declarations

Ethics Approval and Consent to Participate

The study was approved by the Thi-Qar Health Directorate and the administration of Al-Nasiriyah Teaching Hospital (approval No. 264/2025, dated 26 November 2025). Written informed consent was obtained from all participants or their legal guardians before sample collection. The study was conducted in accordance with the Declaration of Helsinki.

Consent for Publication
All authors have given their consent for the publication of this work under the Creative Commons Attribution Non-Commercial 4.0 license.

Data Availability
The datasets generated and analysed during the current study are available from the corresponding author upon reasonable request.

Competing Interests
The authors declare that they have no competing interests.

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Author Contributions
JAA worked on the conceptualisation, investigation, methodology, formal analysis, writing the original draft and visualisation.
HSS worked on the supervision, validation, project administration, writing the review and editing.
Both authors read and approved the final manuscript.

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References

1. Abdelraheem WM, Ismail DE, Hammad SS. Prevalence of bla OXA-48 and other carbapenemase encoding genes among carbapenem-resistant Pseudomonas aeruginosa clinical isolates in Egypt. BMC Infectious Diseases. 2024 Nov 11;24(1):1278. https://doi.org/10.1186/s12879-024-10123-7
2. Alkheeqani B, Khassaf A. Detection and Pathogenicity Features of Pseudomonas Aeruginosa in Patients with Skin Infection. University of Thi-Qar Journal of Science. 2024 Dec 5;11(2):23-9. https://doi.org/10.32792/utq/utjsci/v11i2.1189
3. Gadaime NK, Haddadin RN, Shehabi AA, Omran IN. Antimicrobial resistance and carbapenemase dissemination in Pseudomonas aeruginosa isolates from Libyan hospitals: a call for surveillance and intervention. Libyan Journal of Medicine. 2024 Dec 31;19(1):2344320. https://doi.org/10.1080/19932820.2024.2344320
4. Reem A, Almansoob S, Senan AM, Raj AK, Shah R, Shrewastwa MK, Kumal JP. Pseudomonas aeruginosa and related antibiotic resistance genes as indicators for wastewater treatment. Heliyon. 2024 May 15;10(9). https://doi.org/10.1016/j.heliyon.2024.e29798
5. Chung J, Eisha S, Park S, Morris AJ, Martin I. How three self-secreted biofilm exopolysaccharides of Pseudomonas aeruginosa, Psl, Pel, and alginate, can each be exploited for antibiotic adjuvant effects in cystic fibrosis lung infection. International Journal of Molecular Sciences. 2023 May 13;24(10):8709. https://doi.org/10.3390/ijms24108709
6. Jasim AS, Mohammed AL, Abbas WH, Ibraheim HK, Gharban HA. Identification of blaOXA-23 gene in resistant Pseudomonas aeruginosa strains isolated from cows and humans in Basra province, Iraq. Veterinary World. 2024 Jul 26;17(7):1629. https://doi.org/10.14202/vetworld.2024.1629-1636
7. Magiorakos AP, Srinivasan A, Carey RB, Carmeli Y, Falagas ME, Giske CG, Harbarth S, Hindler JF, Kahlmeter G, Olsson-Liljequist BJ, Paterson DL. Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. Clinical Microbiology and Infection. 2012 Mar 1;18(3):268-81. https://doi.org/10.1111/j.1469-0691.2011.03570.x
8. Kareem SM, kareem Musafer H, Abdulhady ZA, Ali MG. Prevalence of some metallo β-lactamase enzymes genes in P. aeruginosa isolated from different clinical sample in Baghdad, Iraq. Microbial Pathogenesis. 2025 Jan 1;198:107113. https://doi.org/10.1016/j.micpath.2024.107113
9. Hameed HG. Pseudomonas aeruginosa Epidemiology and Antibiotic Resistance: A Five Years Retrospective Study in Iraq. Al-Esraa University College Journal for Medical Sciences. 2023;4(6):31-54. https://doi.org/10.70080/2790-7937.1010
10. Saud A, Hussein RH, Jameel YM. Antibiotic Resistance and Biofilm Formation in Pseudomonas aeruginosa from Different Clinical Samples in Nasiriya, Iraq. University of Thi-Qar Journal of Science. 2025 Jun 27;12(1):216-23. https://doi.org/10.32792/utq/utjsci/v12i1.1342
11. Jassim AA, Mahdi WM, Al-Obaidi OR. Identification of algD and oprL genes in Pseudomonas aeruginosa isolated from multiple environments and different local

sources in Iraq. Kuwait Journal of Science. 2025 Jan 1;52(1):100302. https://doi.org/10.1016/j.kjs.2024.100302
12. Baquero F, Martinez JL, F. Lanza V, Rodríguez-Beltrán J, Galán JC, San Millán A, Cantón R, Coque TM. Evolutionary pathways and trajectories in antibiotic resistance. Clinical Microbiology Reviews. 2021 Dec 15;34(4):e00050-19. https://doi.org/10.1128/CMR.00050-19
13. Venkatesan N, Sikkander S. Microbial biofilms: issues and challenges. Microbial Biofilms. 2023 Jan 1:1-7. https://doi.org/10.1016/B978-0-323-95715-1.00003-0
14. Jihad M, Salih M. Microbial detection and antibiotic susceptibility patterns of clinical isolates from women with urinary tract infection in AL-Nasiriyah city/Iraq. University of Thi-Qar Journal of Science. 2024 Jun 1;11(1):45-54. https://doi.org/10.32792/utq/utjsci/v11i1.1168
15. Zhou J, Tang X, He L, Zhang F, Mou X, Zhao N, Li H, Bao R. Molecular Mechanisms of β-Lactam Resistance and Its Contribution to Multidrug Resistance in Helicobacter pylori. The Journal of Infectious Diseases. 2026 Mar 15;233(Supplement_1):S10-20. https://doi.org/10.1093/infdis/jiaf385
16. Yang S, Li X, Cang W, Mu D, Ji S, An Y, Wu R, Wu J. Biofilm tolerance, resistance and infections increasing threat of public health. Microbial Cell. 2023 Sep 26;10(11):233. https://doi.org/10.15698/mic2023.11.807
17. Mohamed MA, Mohamed HA, Afifi MM. Prevalence of MDR Pseudomonas aeruginosa in Intensive care units and burned patients. Journal of Environmental Studies. 2022 Sep 30;27(1):10-5. https://doi.org/10.21608/jesj.2022.143610.1020
18. Subai Z, Al-Galebi A. Molecular detection of virulence and carbapenem resistance genes in clinical isolates of Pseudomonas Aeruginosa from Iraqi hospitals. Babcock University Medical Journal. 2025 Dec 31;8(2):226-33. https://doi.org/10.38029/babcockuniv.med.j..v8i2.1025
19. Alshamiry M, HF HK. Prevalence of the pelA gene in Pseudomonas aeruginosa and serum IL-22 levels in patients with diabetic foot ulcers. Babcock University Medical Journal. 2025 Dec 31;8(2):451-6. https://doi.org/10.38029/babcockuniv.med.j..v8i2.1138
20. Toth M, Stewart NK, Quan P, Khan MM, Cox J, Buynak JD, Smith CA, Vakulenko SB. Dual mechanism of the OXA-23 carbapenemase inhibition by the carbapenem NA-1-157. Antimicrobial Agents and Chemotherapy. 2025 Oct 1;69(10):e00918-25. https://doi.org/10.1128/aac.00918-25
21. Ma LZ, Wang D, Liu Y, Zhang Z, Wozniak DJ. Regulation of biofilm exopolysaccharide biosynthesis and degradation in Pseudomonas aeruginosa. Annual Review of Microbiology. 2022 Sep 8;76:413-33. https://doi.org/10.1146/annurev-micro-041320-111355
22. Serrato RV. Bacterial Alginate Biosynthesis and Metabolism. InAlginate-Applications and Future Perspectives. IntechOpen. 2022 Dec 23. https://doi.org/10.5772/intechopen.109295
23. Shatti HH, Al-Saeed WM, Nader MI. Effect biofilm formation in Pseudomonas aeruginosa resistance to antibiotic. Mustansiriya Medical Journal. 2022 Jan 1;21(1):13-7. https://doi.org/10.4103/MJ.MJ_11_21