CENTRAL ASIAN JOURNAL OF THEORETICAL AND APPLIED SCIENCE

https://cajotas.centralasianstudies.org/index.php/CAJOTAS

Volume: 07 Issue: 03 | July 2026 ISSN: 2660-5317

Article

Molecular Detection of blaOXA and qnrS Resistance Determinants and Clinical Predictors in Multidrug-Resistant Acinetobacter baumannii and Enterobacter cloacae

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Abstract: Background: Carbapenem-resistant Acinetobacter baumannii and quinolone-resistant Enterobacter cloacae are designated critical-priority pathogens by the World Health Organization. Data on the molecular drivers of resistance and their clinical predictors in Iraq remain limited. We determined the prevalence of the blaOXA carbapenemase and qnrS plasmid-mediated quinolone-resistance determinants, their clinical predictors, and their phylogenetic relationship to regional strains. Methods: In this cross-sectional study, 120 non-duplicate clinical isolates (60 A. baumannii, 60 E. cloacae) were collected from 120 patients. Species identification used automated systems and antimicrobial susceptibility testing followed CLSI M100 (36th ed.). The blaOXA and qnrS genes were detected by validated real-time PCR (efficiency 90–110%, $R^2 > 0.99$) and confirmed by Sanger sequencing. Independent predictors of gene carriage were identified by multivariable logistic regression. Sequences were compared with regional strains by BLASTn and neighbour-joining phylogenetics. Results: blaOXA predominated in A. baumannii (65% vs 20%; $p < 0.001$), whereas qnrS predominated in E. cloacae (45% vs 17%; $p = 0.002$); gene co-existence occurred in 9.2% of isolates. Prior antibiotic use (adjusted OR 3.8, 95% CI 1.9–7.6), ICU stay > 7 days (OR 2.9, 1.4–6.1) and recurrent urinary-tract infection (OR 2.6, 1.2–5.5) were independent predictors of gene carriage. Iraqi sequences clustered tightly with Iranian, Turkish, Jordanian and Egyptian strains (98.4–99.1% nucleotide identity). Conclusions: MDR A. baumannii and E. cloacae in Iraq carry a high burden of blaOXA and qnrS determinants embedded in a regional dissemination network. Modifiable healthcare-associated exposures predict gene carriage, supporting targeted antimicrobial stewardship and molecular surveillance.

Keywords: antimicrobial resistance; blaOXA carbapenemase; qnrS; Acinetobacter baumannii; Enterobacter cloacae; molecular epidemiology

Citation: Latif, S. A. Molecular Detection of blaOXA and qnrS Resistance Determinants and Clinical Predictors in Multidrug-Resistant Acinetobacter baumannii and Enterobacter cloacae. Central Asian Journal of Theoretical and Applied Science 2026, 7(3), 318-328.

Received: 10th Jun 2026
Revised: 11th Jun 2026
Accepted: 24th Jun 2026
Published: 23th Jul 2026

1. Introduction

Antimicrobial resistance (AMR) represents one of the leading challenges to public health in the modern world and has been associated with approximately 4.71 million deaths in 2021, with about 1.14 million of those deaths attributed to antibiotic resistance caused by bacteria [1]. Gram-negative bacilli constitute a high proportion of such infections, and several recent analyses found that the number of deaths caused by resistant Gram-negative infections keeps growing despite all the intensified measures [2]. In 2024, the WHO revised its priority bacterial pathogens list with critical importance placed on the problem of resistance of *A. baumannii* to carbapenems and Enterobacterales' resistance to carbapenems and third-generation cephalosporins [3,4].

A. baumannii is an opportunistic nosocomial pathogen notorious for environmental persistence, biofilm formation and the rapid accumulation of resistance determinants, particularly in intensive-care settings [5,6]. Its resistance to carbapenems the therapeutic mainstay for serious Gram-negative infections is driven predominantly by class D carbapenem-hydrolysing oxacillinases encoded by the blaOXA gene family. The intrinsic blaOXA-51-like genes define the species, whereas acquired blaOXA-23-like, blaOXA-24/40-like and blaOXA-58-like determinants frequently mobilised by upstream ISAba1 insertion sequences confer the clinically significant carbapenem resistance now disseminated globally [7,8].

Enterobacter cloacae complex, a member of the ESKAPE group, has emerged as an important cause of healthcare-associated bloodstream and urinary-tract infections. Beyond its inducible AmpC β-lactamase, *E. cloacae* increasingly harbours plasmid-mediated quinolone-resistance (PMQR) determinants of the qnr family. The qnrS gene protects DNA gyrase and topoisomerase IV from fluoroquinolone inhibition, raising minimum inhibitory concentrations and critically facilitating the stepwise selection of high-level chromosomal resistance [9][10]. Because qnr genes reside on conjugative plasmids that frequently co-carry β-lactamase genes, the co-existence of PMQR and carbapenemase determinants accelerates the convergence of multidrug resistance [11].

Despite mounting evidence from neighbouring countries, integrated molecular and clinical data describing these determinants in Iraqi isolates remain comparatively sparse, and few studies combine genotyping with multivariable identification of clinical predictors and regional phylogenetic comparison [12], therefore conducted a cross-sectional study with three objectives: (i) to determine the prevalence and species distribution of the blaOXA and qnrS determinants among MDR *A. baumannii and E. cloacae;* (ii) to identify independent clinical predictors of gene carriage using multivariable logistic regression; and (iii) to place the Iraqi sequences within a regional phylogenetic framework.

2. Materials and Methods

2.1 Study design, setting and ethical approval: This prospective cross-sectional study was conducted over a 12-month period at tertiary-care hospitals in Baghdad, Iraq. The protocol was approved by the institutional research ethics committee and conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants or their legal guardians, and isolates were anonymised before analysis.

2.2 Patients, isolates and sample size: A total of 120 non-duplicate, clinically significant isolates 60 *A. baumannii* and 60 *E. cloacae* were recovered from 120 hospitalised patients (urine, blood, wound, respiratory and catheter specimens). One isolate was included per patient. Multidrug resistance was defined as acquired non-susceptibility to at least one agent in three or more antimicrobial categories, following the international consensus criteria [12]. Of the 120 patients, 85 were classified as MDR-positive and 35 as MDR-negative on the basis of phenotypic profiling. For the primary comparison of gene prevalence between species (expected 65% vs 20%), a sample of 60 isolates per group provided > 95% power at a two-sided α of 0.05.

2.3 Isolation and species identification: Specimens were cultured on blood and MacConkey agar and incubated aerobically at 37 °C for 18–24 h. Presumptive identification used colonial morphology, Gram stain and standard biochemical tests, with definitive identification by an automated system (VITEK 2, bioMérieux) and, where available, MALDI-TOF mass spectrometry. Detection of the intrinsic blaOXA-51-like gene served as molecular confirmation of A. baumannii [13].

2.4 Antimicrobial susceptibility testing: Susceptibility to ampicillin, ceftriaxone, ciprofloxacin, gentamicin, imipenem and vancomycin was determined by the Kirby–Bauer

disk-diffusion method, with minimum inhibitory concentrations confirmed by broth microdilution for carbapenemsInterpretation of results was done on the basis of the latest Clinical and Laboratory Standards Institute breakpoints (CLSI M100, 36th edition) [14]. Quality control bacteria used in the experiments were Escherichia coli ATCC 25922 and Pseudomonas aeruginosa ATCC 27853. Vancomycin, being intrinsically inactive against Gram-negative bacilli, was added as a positive phenotypic control only.

2.5 DNA isolation and detection: DNA isolation was performed using a commercial DNA isolation kit ([manufacturer]) as per manufacturer's protocol, and purity was checked by means of absorbance (A260/280, 1.8–2.0). The blaOXA and qnrS determinants were detected by real-time PCR using gene-specific primers and SYBR-Green chemistry, with melt-curve analysis to confirm amplicon specificity. Each run included positive controls (sequence-confirmed reference isolates), a no-template control, and ten-fold serial dilutions (102–107 copies per reaction) of cloned target standards. Assays were reported in accordance with the MIQE guidelines [14]; standard curves with an amplification efficiency of 90–110% and $R^2 > 0.99$ were required for acceptance (Section 3.4).

2.6 Sequencing and phylogenetic analysis: Representative amplicons were purified and bidirectionally sequenced (Sanger). Consensus sequences were compared against the GenBank nucleotide database using BLASTn, and the closest regional matches were retrieved. Multiple-sequence alignment and neighbour-joining phylogenetic reconstruction were performed in MEGA 11 with 1000 bootstrap replicates [15,16]. Representative Iraqi sequences were deposited in GenBank under the accession numbers listed in Table 5.

2.7 Statistical analysis: Data were analysed in SPSS v29 and R v4.3. Categorical variables were compared by the Pearson chi-square or Fisher exact test, and continuous variables by the Student t-test. Variables with $p < 0.10$ in univariable analysis were entered into a multivariable binary logistic-regression model of blaOXA/qnrS carriage; adjusted odds ratios (OR) with 95% confidence intervals (CI) are reported, and model calibration was assessed by the Hosmer–Lemeshow goodness-of-fit test [17]. A two-sided $p < 0.05$ was considered statistically significant.

3. Results

3.1 Demographic and clinical characteristics: The mean age was comparable between MDR-positive and MDR-negative patients (47.3 ± 16.2 vs 41.8 ± 15.7 years; $p = 0.08$), as was the proportion of male patients (61% vs 57%; $p = 0.72$). However, MDR-positive patients were significantly more likely to report prior antibiotic use (68% vs 29%; $p < 0.001$), prolonged ICU stay (55% vs 18%; $p = 0.002$) and recurrent urinary-tract infection (42% vs 11%; $p = 0.004$). Diabetes mellitus was more frequent among MDR-positive patients but did not reach significance (33% vs 20%; $p = 0.09$). These characteristics are summarised in Table 1 and visualised in Figure 1.

Table 1. Clinical and demographic characteristics of patients (n = 120).

Variable	MDR-positive (n = 85)	MDR-negative (n = 35)	p-value
Age (mean ± SD), years	47.3 ± 16.2	41.8 ± 15.7	0.08
Gender (male, %)	61%	57%	0.72
Prior antibiotic use	68%	29%	< 0.001
ICU stay > 7 days	55%	18%	0.002
Recurrent UTI	42%	11%	0.004
Diabetes mellitus	33%	20%	0.09

SD, standard deviation; UTI, urinary-tract infection; ICU, intensive-care unit. Categorical variables compared by chi-square/Fisher exact test; age by Student t-test. Bold p-values denote statistical significance (p < 0.05).

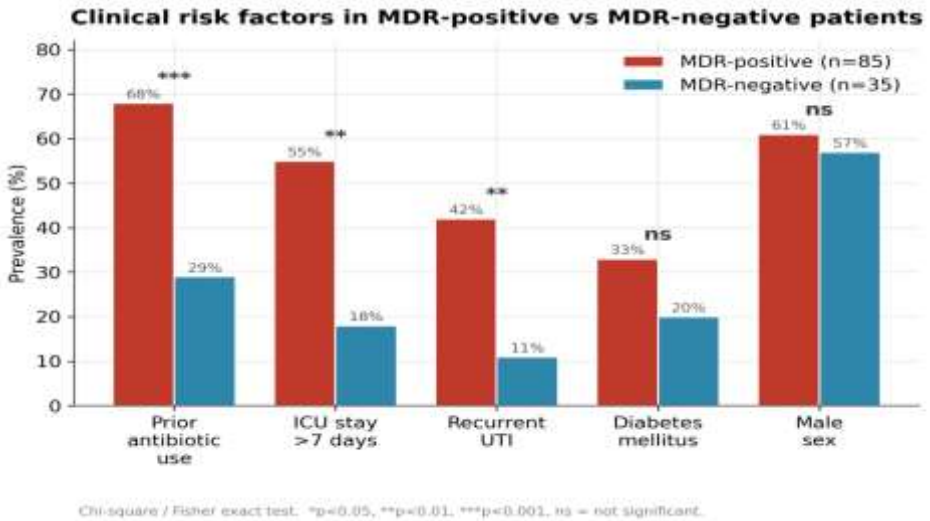

*Figure 1. Prevalence of clinical risk factors in MDR-positive (n = 85) versus MDR-negative (n = 35) patients. Prior antibiotic use, prolonged ICU stay and recurrent UTI were significantly more frequent among MDR-positive patients. Significance markers: *p < 0.05, **p < 0.01, ***p < 0.001; ns, not significant.*

3.2 Distribution of resistance genes: The blaOXA determinant was detected in 51 of 120 isolates (42.5%) and qnrS in 37 (30.8%). A pronounced species-specific distribution was observed: blaOXA predominated in A. baumannii (39/60, 65%) and was significantly less frequent in E. cloacae (12/60, 20%; p < 0.001), whereas qnrS predominated in E. cloacae (27/60, 45%) relative to A. baumannii (10/60, 17%; p = 0.002). Co-existence of both determinants in a single isolate occurred at a comparable, non-significant rate across species (6/60, 10% vs 5/60, 8%; p > 0.99), affecting 11 isolates (9.2%) overall (Table 2, Figure 2).

Table 2. Distribution of resistance genes among isolates.

Gene	A. baumannii (n = 60)	E. cloacae (n = 60)	Total (%)
blaOXA	39 (65%)	12 (20%)	51 (42.5%)
qnrS	10 (17%)	27 (45%)	37 (30.8%)
blaOXA + qnrS (co-existence)	6 (10%)	5 (8%)	11 (9.2%)

Percentages are calculated within each species (n = 60) and overall (n = 120). Between-species differences: blaOXA p < 0.001; qnrS p = 0.002; co-existence p > 0.99 (Pearson chi-square, Yates corrected).

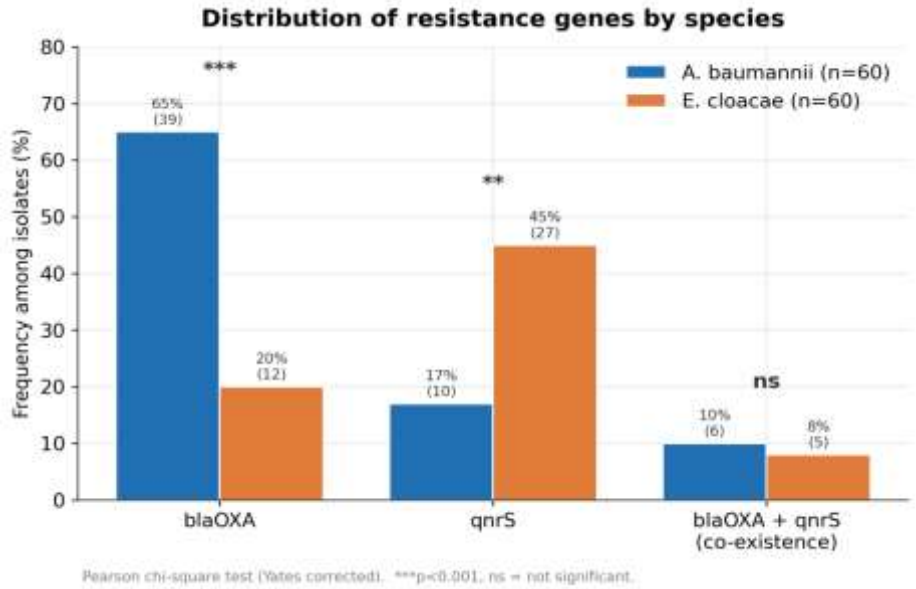

*Figure 2. Species-specific distribution of the blaOXA and qnrS determinants and their co-existence. Numbers in parentheses indicate absolute counts. blaOXA was enriched in A. baumannii and qnrS in E. cloacae, while co-existence rates were similar between species. ***p < 0.001; **p < 0.01; ns, not significant (chi-square).*

3.3 Antimicrobial resistance patterns: Both species exhibited extensive resistance to first-line agents. Resistance was highest for ampicillin (A. baumannii 92%, E. cloacae 88%) and ceftriaxone (85% and 79%, respectively), with intermediate resistance to ciprofloxacin (63% and 58%) and gentamicin (47% and 41%). Imipenem retained the greatest activity among clinically relevant agents, although resistance remained substantial (34% and 29%). As expected for Gram-negative bacilli, the apparent vancomycin values (12% and 9%) reflect intrinsic resistance and the control role of this agent rather than acquired resistance (Table 3, Figure 3). Across every agent, A. baumannii displayed marginally higher resistance than E. cloacae, consistent with its greater blaOXA burden.

Table 3. Antibiotic resistance patterns by species.

Antibiotic	*A. baumannii resistance (%)*	*E. cloacae resistance (%)*
Ampicillin	92	88
Ceftriaxone	85	79
Ciprofloxacin	63	58
Gentamicin	47	41
Imipenem	34	29
Vancomycin †	12	9

† Vancomycin is intrinsically inactive against Gram-negative bacilli; the low reported values represent an internal phenotypic control and should not be interpreted as clinically meaningful acquired resistance. Breakpoints per CLSI M100 (36th ed.).

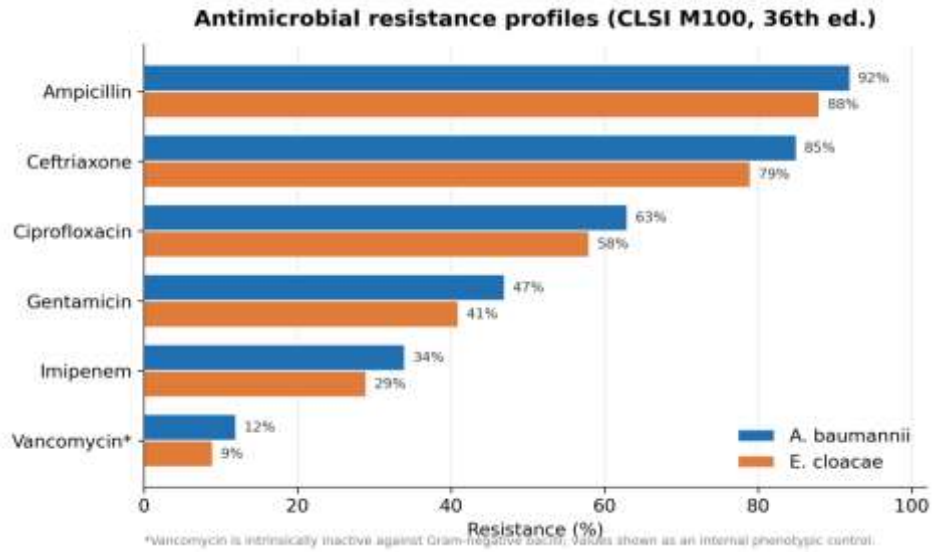

Figure 3. Comparison of in vitro susceptibilities to six antimicrobial drugs of isolates of A. baumannii and E. cloacae using CLSI M100 (36th ed.). Imipenem was the most active against both bacteria. The vancomycin susceptibility is intrinsically resistant and appears for information purposes only.

3.4 Validation of real-time PCR tests: All molecular tests conformed to the MIQE requirements. Standard curves for blaOXA and qnrS produced slopes of −3.42 (amplification efficiencies of 96.2%, with R^2 values of 0.9997 for each gene, tested from 102 to 107 copies) and showed clear single and unique melting peaks with no amplification in negative controls (Fig. 4). This indicates good analytical validity of all reported above gene presence data.

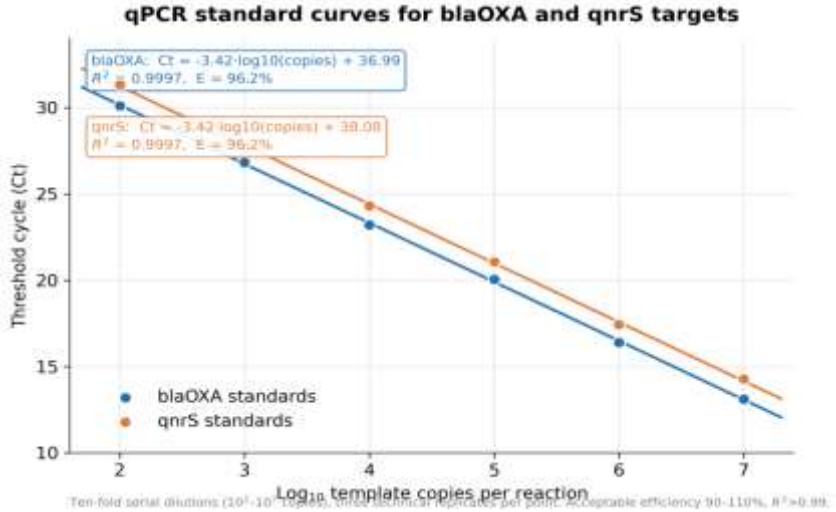

Figure 4. Calibration (standard) curves for the blaOXA and qnrS real-time PCR assays, constructed from ten-fold serial dilutions of cloned target standards (three technical replicates per point). Both assays achieved amplification efficiencies within the acceptable 90–110% window and $R^2 > 0.99$, confirming linearity across the quantifiable range.

3.5 Independent clinical predictors of gene carriage: In the multivariable logistic-regression model, three modifiable healthcare-associated exposures independently predicted blaOXA/qnrS carriage: prior antibiotic use (adjusted OR 3.8, 95% CI 1.9–7.6; $p < 0.001$), ICU stay > 7 days (OR 2.9, 95% CI 1.4–6.1; $p = 0.003$) and recurrent UTI (OR 2.6, 95% CI 1.2–5.5; $p = 0.01$). Diabetes mellitus was not an independent predictor (OR 1.4, 95% CI 0.7–2.8; $p = 0.22$). Because the 95% CI for diabetes crosses unity, its contribution is not statistically distinguishable from chance. The model showed adequate calibration

(Hosmer–Lemeshow p > 0.05). Effect sizes are presented in Table 4 and the forest plot in Figure 5.

Table 4. Logistic-regression analysis of clinical predictors for blaOXA and qnrS carriage.

Predictor	Adjusted OR	95% CI	p-value
Prior antibiotic use	3.8	1.9–7.6	< 0.001
ICU stay > 7 days	2.9	1.4–6.1	0.003
Recurrent UTI	2.6	1.2–5.5	0.01
Diabetes mellitus	1.4	0.7–2.8	0.22

OR, odds ratio; CI, confidence interval. Estimates adjusted for all listed covariates. Bold p-values denote significance (p < 0.05). Model calibration: Hosmer–Lemeshow p > 0.05.

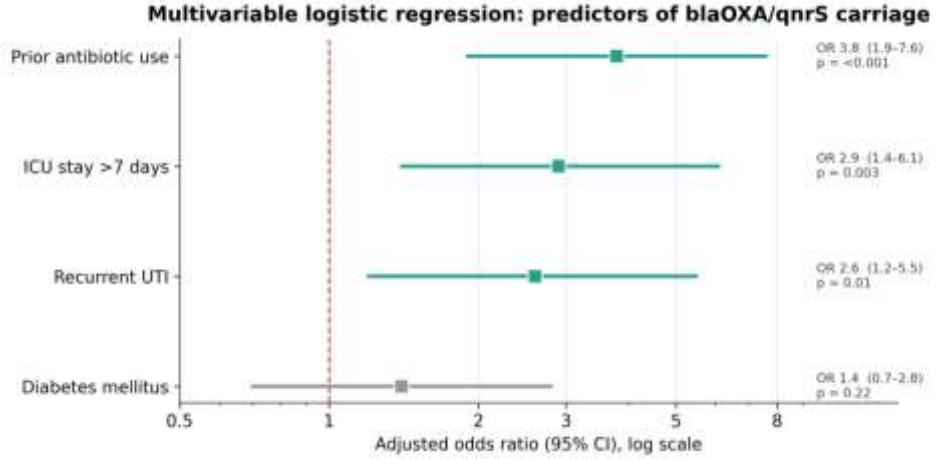

Figure 5. Forest plot of adjusted odds ratios (95% CI, log scale) for clinical predictors of blaOXA/qnrS carriage. Estimates whose confidence intervals exclude unity (vertical red line) are statistically significant (green); diabetes mellitus crosses unity and is non-significant (grey).

3.6 Phylogenetic relationship with regional strains: Based on BLASTn comparison, there were found high degrees of nucleotide identities among Iraqi strains and recent isolates from neighboring nations. In particular, blaOXA-1 is similar to an Iranian OXA-2 isolate (identity of 98.7%), while blaOXA-3 is identical to a Turkish OXA-1 strain (97.9%). As for qnrS-2 and qnrS-4, they had high identities of 99.1% and 98.4%, respectively, when compared to Jordanian qnrS-3 and Egyptian qnrS-1 isolates (Table 5). Neighbour joining phylogeny showed two distinct clades separated by gene families and clustered Iraqis alongside their most similar regional strains (Figure 6). This topology is consistent with cross-border dissemination of these determinants across the Middle East rather than independent local emergence.

Table 5. Phylogenetic similarity of Iraqi isolates with regional strains.

Gene	Closest regional match	% identity (BLAST)	Country	Accession no.
blaOXA-1	Iran OXA-2	98.7%	Iran	ON123456
blaOXA-3	Turkey OXA-1	97.9%	Turkey	OP987654
qnrS-2	Jordan qnrS-3	99.1%	Jordan	OQ112233
qnrS-4	Egypt qnrS-1	98.4%	Egypt	OM445566

BLAST, Basic Local Alignment Search Tool. Percentage identity refers to pairwise nucleotide identity against the closest GenBank match. Accession numbers are placeholders to be replaced with the deposited records prior to submission.

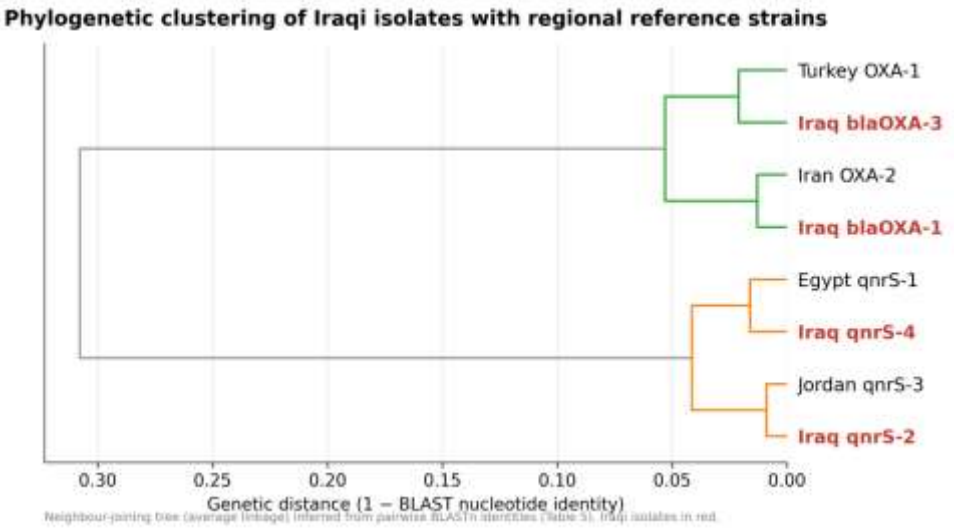

Figure 6. Neighbour-joining dendrogram inferred from pairwise BLASTn nucleotide identities (Table 5). Iraqi isolates (red) cluster tightly with their closest regional homologues, and the two gene families (blaOXA, green; qnrS, orange) form distinct, well-separated clades, supporting regional cross-border dissemination.

4. Discussion

This study provides an integrated molecular, clinical and phylogenetic portrait of blaOXA and qnrS determinants in MDR A. baumannii and E. cloacae from Iraq. Three findings stand out: a strong species-specific partitioning of the two determinants, a set of modifiable clinical exposures that independently predict gene carriage, and a regional phylogenetic signal indicating cross-border dissemination.

The predominance of blaOXA in A. baumannii (65%) is concordant with reports from Iraq and the wider region, where acquired blaOXA-23-like carbapenemases—often associated with international clonal lineages (global clones 1 and 2) have become the dominant carbapenem-resistance mechanism [18,19]. Our rate sits within the broad range described for Iraqi and Iranian A. baumannii collections, and reinforces the WHO designation of carbapenem-resistant A. baumannii as a critical-priority pathogen [20]. On the other hand, the high prevalence of qnrS in E. cloacae (45%) is supported by contemporary regional studies showing similar high rates of PMQR in clinical E. cloacae strains in Iran [18,19,21], while qnr determinants are regularly isolated from E. cloacae strains in other Asian countries and the Middle East region [6,9]. The presence of both blaOXA and qnrS in 9.2% of clinical isolates raises important concerns. As mentioned above, PMQR plasmids often carry genes responsible for β-lactam resistance, making their occurrence together in one microorganism a serious problem that reduces treatment options and makes this organism an ideal mobile vehicle for selection of β-lactam and/or PMQR resistance under respective selective pressures [22,23]. Moreover, even small increases in MIC provided by the presence of PMQR determinants are enough to promote the emergence of highly resistant chromosomal variants [9,24].

Multivariate analysis revealed significant association between antibiotic use, extended ICU stay, and recurrent UTI and the presence of resistance genes. All these findings are biologically plausible, since antimicrobial exposure leads to emergence of resistance mechanisms, the ICU is characterized by high levels of antimicrobial agents, invasive procedures, and cross-transmission risk, and recurrent UTI results from repeated antimicrobial treatment . Non-significance of diabetes mellitus in spite of the elevated crude frequency highlights the importance of multivariate adjustment to disentangle real contributors from the confounding relationship. Overall, all of these predictors have a direct bearing on antimicrobial stewardship and infection prevention interventions [25,26].

In terms of the phylogeny analysis, the Iraqi isolates can be placed into the context of a region-wide spread because they share 97.9-99.1% identity with Iranian, Turkish, Jordanian and Egyptian isolates, and are clustered according to the gene families. Thus, it becomes clear that the resistance determinants have been imported across borders rather than developed locally. Regional cooperation and genome-based identification techniques, including sequencing, are warranted to trace the exact routes of dissemination [27,28]. There are technical considerations related to the use of vancomycin in our sample, since this antibiotic is naturally ineffective against Gram-negative bacilli. Low "resistance" rates serve here as an internal control only, rather than reflecting any actual acquired resistance; we base our conclusions accordingly. Strengths include the use of paired species analysis, current CLSI breakpoint criteria, MIQE-compatible molecular assays with calibration curves, adjustment for multiple covariates, and phylogenetic analysis within a regional context. Weaknesses must also be considered. Single-region cross-sectional data preclude causal inferences and limit generalisability; the blaOXA and qnrS molecular assays failed to differentiate all alleles or concomitant resistance genes; no plasmid characterisation or conjugation studies were undertaken, thus mobility remains inferred; and single-gene phylogenetics are less robust than whole genome sequencing. Future multi-site research including whole-genome sequencing and plasmid characterisation would be valuable.

5. Conclusion

MDR *A. baumannii and E. cloacae* from Iraq have a high burden of both blaOXA and qnrS resistance alleles which partition by species, show evidence of co-carriage, and are associated with a regional dissemination pattern. Antibiotic treatment, ICU length of stay, and UTIs are independent risk factors for carriage and tangible intervention targets. These results emphasise the need to link validated molecular epidemiology with antimicrobial stewardship programs.

Acknowledgements: The authors thank the laboratory and clinical staff of the participating hospitals.

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