

Antibiotic Resistance and Detection of Qnr Genes in isolated Uropathogenic Bacteria from Patients with Urinary Catheters

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Abstract Antibiotic resistance in bacteria is a serious global public health problem. This study aimed to characterize antibiotic resistance genes in patients with urinary catheters. The study involved bacterial strain collection, antibiotic susceptibility testing, and molecular characterization for the detection of genes mediated by fluoroquinolone resistance. A total of 81 strains were collected. Strain distribution showed that *Staphylococcus aureus* was the most prevalent species (44.44%, n=36). Resistance profiling showed that coagulase-negative *Staphylococcus* strains (n=12) and *Acinetobacter baumannii* strains (n=5) expressed 100% resistance to cefoxitin and all tested beta-lactam antibiotics, respectively. *Acinetobacter baumannii* was resistant to ciprofloxacin (100%, n=5). However, amikacin showed greater activity against *Pseudomonas aeruginosa* (n=28) and *Acinetobacter baumannii* (n=5) strains, with 25% and 0% activity, respectively. Molecular gene characterization identified the qnr A, qnr B, and qnr S genes. The strains exhibited a high diversity of resistance genes, with 12.5%, 77.5%, and 60% observed in qnr A, qnr B, and qnr S, respectively. Co-expression of fluoroquinolone resistance genes was demonstrated in 30 strains. This study highlights the high prevalence of antibiotic-resistant bacteria and underscores the crucial role of microbiological and molecular surveillance of bacteria. The results highlight the need for rigorous antibiotic management to prevent the spread of resistant strains.

Keywords: Antibiotic resistance, uropathogens, urinary catheter, co-expression

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1. Introduction

Antibiotic resistance is a major threat to global public health today. It is defined as the ability of bacteria to survive and multiply despite the presence of antibiotics, drugs designed to inhibit or eliminate them. This phenomenon, naturally present in bacterial evolution, has been greatly amplified by the excessive and often inappropriate use of antibiotics in human and veterinary medicine, as well as in agriculture [1].

Among antibiotics are fluoroquinolones. They represent an important class of antibiotics used to treat a wide range of bacterial infections, including urinary, respiratory, and gastrointestinal infections. However, resistance to fluoroquinolones expressed by certain bacteria has developed rapidly, compromising their clinical effectiveness. These antibiotics target bacterial enzymes

essential for DNA replication, namely DNA gyrase and topoisomerase IV. Point mutations in the genes encoding these enzymes, particularly *gyrA* and *parC*, reduce the bactericidal effect of these drugs [2]. These mutations in quinolone-determining regions (QRDRs) are the main resistance markers observed in many bacterial species.

Furthermore, resistance can also result from increased activity of membrane efflux systems, which actively expel fluoroquinolones from the bacterial cell [3]. In addition, alterations in membrane permeability can limit the entry of fluoroquinolones into the cell, contributing to resistance [4]. The clinical impact of these markers is significant. A French study reported a notable increase in fluoroquinolone resistance in *Escherichia coli* isolated from urinary tract infections between 2015 and 2019, reflecting the spread of QRDR mutations and efflux mechanisms [5]. This trend underscores the importance of monitoring resistance markers to adapt therapeutic strategies and limit the spread of resistant strains.

Fluoroquinolone resistance, mediated by *qnr* genes, is a growing public health problem in West Africa. A study conducted in Nigeria found that 93.3% of fluoroquinolone-resistant *Escherichia coli* isolates carried at least one *qnr* resistance gene, with rates of 33.3% for *qnrA* and 60% for *qnrB*, respectively. No *qnrS* genes were detected in this study [6].

In Gabon, a study demonstrated high resistance to quinolones in enteric pathogens isolated from the stool of young children, with resistance rates of 100% to ofloxacin and 97.3% to ciprofloxacin. The *qnrB* and *qnrS* genes were detected in 13.5% and 13.5% of the strains, respectively, while the *qnrA* gene was not identified [7]. In Côte d'Ivoire, several studies have highlighted the presence of these resistance genes. Research conducted in Abidjan detected the *qnrD* (36.7%), *qnrB* (36.7%), *qnrS* (13.3%), and *qnrA* (6.7%) genes in Gram-negative bacilli. Notably, this study was the first to identify the *qnrD* gene in certain Ivorian bacterial strains [8].

Thus, antibiotic resistance leads to a significant increase in morbidity and mortality rates, lengthens hospital stays, and raises healthcare costs. Furthermore, it limits available treatment options, rendering some treatments ineffective, which poses a real challenge to healthcare systems worldwide [4].

Faced with this crisis, comprehensive strategies are essential. These include more rational use of antibiotics, the development of new drugs, and the strengthening of hygiene and infection control measures. The objective of this study is to characterize the *qnr A*, *B*, and *S* genes in uropathogenic bacteria isolated from urinary catheter users.

2. Materials and Methods

2.1. Collection of Uropathogenic Strains

The bacteria were collected from the Biocollection of the National Reference Center for Antibiotics (CNR) at the Pasteur Institute of Côte d'Ivoire (IPCI). A total of 81 bacterial strains, consisting of *Staphylococcus aureus* (36), *Pseudomonas aeruginosa* (28), coagulase-negative *Staphylococcus* (12), and *Acinetobacter baumannii* (5), were isolated from the urine of patients with urinary catheters during 2022. These isolates were stored at -80°C in a 1% glycerol-enriched brain broth at the IPCI's Biological Resource Center (CeReB). The bacterial strain identifiers were determined based on information related to the hospital department (nephrology, outpatient, neurology, intensive care, pediatrics, pulmonology, emergency), the patient's age, and sex.

2.2. Revivification of Bacterial Strains

Using a Pasteur pipette, the strains were collected and cultured in nutrient broth, Heart and Brain Broth (BCC), and then incubated at 37°C for 24 h. Next, they were successively inoculated onto the selective medium (Chapman, Cetrinide, and Eosin Methylene Blue (EMB) agar) contained in Petri dishes. The dishes were then

incubated at 37°C for 24 h to obtain young bacterial colonies, which were then subcultured onto ordinary agar for antibiotic susceptibility testing.

2.3. Determination of the Phenotype of Collected Strains to Antibiotics

The method of scattering antibiotic-impregnated discs onto Müller-Hinton agar was used to evaluate the susceptibility of the strains according to the recommendations of the Antibiogram Committee of the French Society for Microbiology [9]. The antibiotic-impregnated discs used are listed in Table 1.

Table 1. List of tested antibiotic-impregnated discs

Families of antibiotics	Antibiotics tested	Abbreviations	Disk charges (µg)
Beta-lactams	Ticarcillin	TIC	75
	Cefepime	FEP	30
	Ceftazidime	CAZ	10
	Cefoxitin	FOX	30
	Imipenem	IMP	10
	Meropenem	MEM	10
	Ticarcillin/Clavulanic Acid	TCC	75/10
Fluoroquinolones	Aztreonam	ATM	30
	Ciprofloxacin	CIP	5
	Norfloxacin	NOR	5
Aminosides	Moxifloxacin	MOX	5
	Gentamicin	GEN	10
Macrolides	Amikacin	AKN	30
Lincosamides	Erythromycin	ERY	15
	Clindamycin	CMN	2

2.4. Detection of Qnr Genes by PCR

DNA extraction from the collected strains and the reference strains *Escherichia coli* ATCC 29522 and *Klebsiella pneumoniae* ATCC 70603 was performed using alkaline lysis with phenolization. Conventional polymerase chain reaction (PCR) was used to detect fluoroquinolone resistance genes (*qnr A*, *B*, and *S*). Specific primer pairs listed in Table 2 were used to amplify the genes. PCR amplification was performed in a 25 µL volume using a thermocycler (Perkin® Elmer Gen Amp Lapplied Biosystems 9700). The amplification conditions consisted of an initial DNA denaturation step for 5 min at 95°C. This step was followed by 35 amplification cycles, including denaturation at 95°C for 1 min, hybridization at 56°C for 1 min, elongation at 72°C for 1 min 30 s, and a final elongation step at 72°C for 10 min. The reaction medium consisted of 12.5 µL of Master Mix, 1 µL of forward and antisense primers, and 6.5 µL of ultrapure water. To each well of the PCR plate, pre-filled with the mixture, 5 µL of bacterial DNA was added. Another reaction mixture without DNA was used as the negative control. The amplified products were analyzed by electrophoresis on 1.5% agarose gel (Invitrogen) stained with ethidium bromide. The reading was taken on an ultraviolet (UV) plate (Gel Doc).

Table 2. List of primers used for the detection of fluoroquinolone resistance genes

Gene name	Primer	5'-3' primer sequences	Amplicons (pb)	References
<i>qnr A</i>	F	GATAAAGTTTTCAGCAAGAGG	542	Touati <i>et al.</i> , 2008
	R	ATCCAGATCGGCAAAGGTT		
<i>qnr B</i>	F	GACAGAAACAGGTTACCG GT	594	Touati <i>et al.</i> , 2008
	R	CAAGACGTTCCAGAGCAA CG		
<i>qnr S</i>	F	GACGTGCTAACTTGCGTGAT	388	Seyed <i>et al.</i> , 2014
	R	AACACCTCGACTTAAGTCTGA		

3. Results

3.1. Distribution of Collected Strains According to Hospital Departments

Analysis of the results showed that the nephrology department had the highest isolation rate at 60.49%, followed by the outpatient department (13.58%). The lowest bacterial rate (1.23%) was recorded in the surgery and intensive care departments ($\chi^2 = 51.87$; $p = 0.000$). The complete dataset is shown in Figure 1.

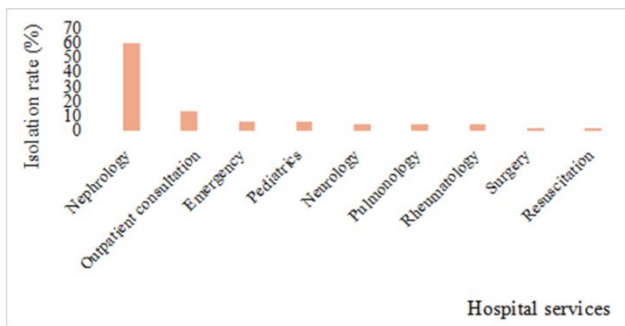


Figure 1. Distribution of collected strains according to hospital departments

3.2. Distribution of Strains According to Sex and Age of Patients

Of the 81 bacterial strains, 69 were isolated from males (85%) and 12 from females (15%), for a sex ratio of 5.75. Analysis revealed a predominance of strains isolated from males compared to females ($\chi^2 = 24.60$; $p = 0.000$). Regarding age, the analysis showed a significant difference in isolation rates across age groups ($\chi^2 = 14.29$; $p = 0.003$). The most represented age group was that of elderly patients aged 51 to 73 years (46%), followed by the 36-50 year age group (25%). The lowest rate of isolation (9%) was represented by individuals aged 0 to 18 years (Figure 2).

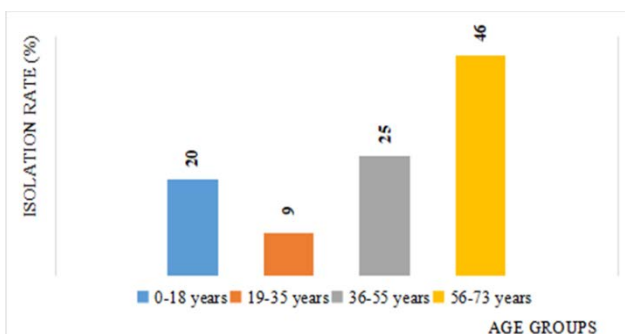
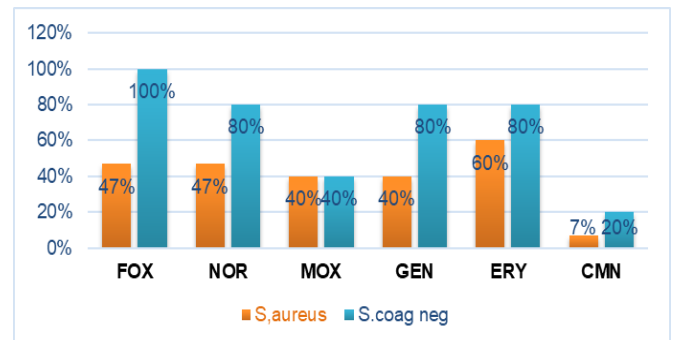


Figure 2. Distribution of collected strains according to age class

3.3. Antibiotic Resistance Profile of Collected Strains

Resistance of *Staphylococcus aureus* and coagulase-negative *Staphylococcus* to cefoxitin, erythromycin, and clindamycin

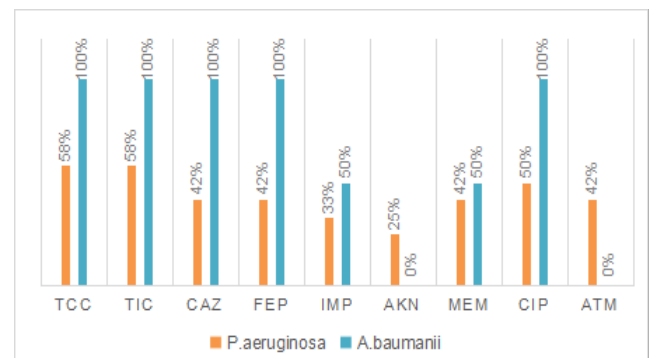
Cefoxitin was the only beta-lactam molecule tested in these species. Of the 36 *Staphylococcus aureus* strains tested, 17 were resistant, representing a resistance rate of 47.22%. In contrast, all 12 coagulase-negative *Staphylococcus* strains tested were completely resistant (100%). In this study, the results showed that in the presence of gentamicin and erythromycin, both *Staphylococcus aureus* and coagulase-negative *Staphylococcus* exhibited a low resistance rate (40%). Furthermore, coagulase-negative *Staphylococcus* showed resistance rates of 80% and 20% to erythromycin and clindamycin, respectively (Figure 3).



FOX: cefoxitin; NOR: norfloxacin; MOX: moxifloxacin; GEN: gentamicin ERY: erythromycin CMN: clindamycin

Figure 3. Resistance rates of staphylococci to antibiotics

Resistance of *Pseudomonas aeruginosa* and *Acinetobacter baumannii* to beta-lactams



TCC: ticarcillin (clavulanic acid); TIC: ticarcillin; CAZ: ceftazidime; FEP: cefepim; IMP: imipenem; AKN: amikacin; MEN: meropenem; CIP: ciprofloxacin; ATM: aztreonam

Figure 4. Resistance rate of non-fermenting Gram-negative bacilli to antibiotics

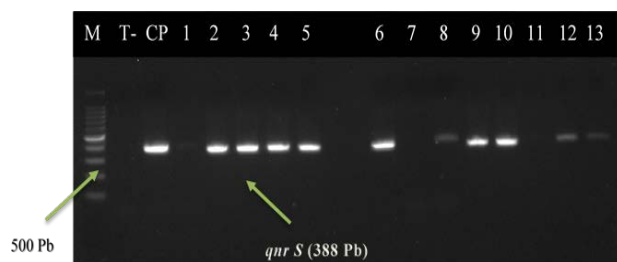
A total of 33 strains, including 5 *Acinetobacter baumannii* and 28 *Pseudomonas aeruginosa*, were tested against beta-lactams. Each of these species showed different resistance phenotypes in the presence of the antibiotics used. *Acinetobacter baumannii* exhibited high resistance rates (100%) to almost all the beta-lactams tested, except for imipenem, to which the bacteria were partially resistant (50%). As for *Pseudomonas aeruginosa*, the results showed a resistance rate of 58% against ticarcillin + clavulanic acid and ticarcillin alone, compared to 42% for cefepime, ceftazidime, imipenem, and meropenem (Figure 4).

Germ resistance to fluoroquinolones

Resistance rates of 47% and 60% for norfloxacin and moxifloxacin were observed in the presence of *Staphylococcus aureus*. In contrast, coagulase-negative staphylococci showed a high resistance rate of 80% to these antibiotics. Regarding *Acinetobacter baumannii* and *Pseudomonas aeruginosa* strains, resistance rates of 100% and 50% were observed against ciprofloxacin (Figure 4).

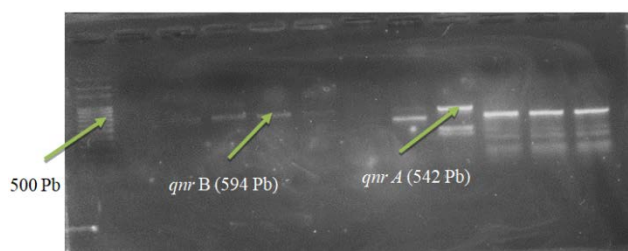
3.4. Genotypes of the Studied Strains

Fluoroquinolone Resistance Genes Detected in the Tested Strains



Lane M: molecular weight marker (Solis Biodyne 100 Pb), Lane CP: positive control, Lane T: negative control, Lanes 7 and 11: negative samples, Lanes 1, 2, 3, 4, 5, 6, 8, 9, 10, 12, 13: positive samples.

Figure 5. Electrophoretic profiles on 1.5% agarose gel of the qnr S gene detected in the tested strains



Lane M: molecular weight marker (Solis Biodyne 100 Pb), Lane CP: positive control, Lane T: negative control, Lane 5: negative samples, Lanes 1, 2, 3, 4, 6, 7: positive samples for qnr B, Lanes 8, 9, 10: positive samples for qnr A.

Figure 6. Electrophoretic profiles on 1.5% agarose gel showing multiplex PCR for the detection of the qnr A and qnr B genes

A total of 40 strains, consisting of *Staphylococcus aureus* (n=17), *Pseudomonas aeruginosa* (n=13), coagulase-negative *Staphylococcus* (n=8), and *Acinetobacter baumannii* (n=2), phenotypically resistant to ciprofloxacin, norfloxacin, and moxifloxacin, were selected for molecular analysis. Analysis of the results revealed that resistance genes were present at varying levels. Specifically, the qnrB and qnrS genes were

predominant, with levels of 80% and 62.5%, respectively, in 32 and 25 strains. The qnrA gene was present in 5 strains at a level of 12.5%. Figure 5 and Figure 6 present the electrophoretic profiles of the genes analyzed.

Simultaneous Expression of Fluoroquinolone Resistance Genes

Agarose gel electrophoresis revealed the simultaneous expression (co-expression) of fluoroquinolone resistance genes. Indeed, the *Acinetobacter baumannii* strains expressed the different types of co-expression: qnr A-B, qnr B-S, and qnr A-B-S, with levels of 50% (n=1), 100% (n=2), and 50% (n=1), respectively. However, *Staphylococcus aureus*, coagulase-negative *Staphylococcus*, and *Pseudomonas aeruginosa* exhibited only one type of co-expression, qnr B-S. Table 3 presents all the results.

Table 3. Distribution of fluoroquinolone resistance genes detected in the tested strains

Souches (N)	Genes N (%)			Co-expression N (%)		
	qnr A	qnr B	qnr S	qnr A-B	qnr B-S	qnr A-B-S
<i>S. aureus</i> (17)	3(17, 64)	16 (94,11)	8 (47,06)	0	7 (41,17)	0
<i>S. coag neg</i> (08)	0	4 (50)	4 (50)	0	4 (50)	0
<i>P. aeruginosa</i> (13)	0	10 (76,92)	11 (84,61)	0	7 (53,84)	0
<i>A. baumannii</i> (02)	2 (100)	2 (100)	2 (100)	1 (50)	2 (100)	1 (50)
Total (40)	5	32	25	1	20	1

S. aureus: *Staphylococcus aureus* ; *S. coag neg*: coagulase negative *Staphylococcus*; *P. aeruginosa*: *Pseudomonas aeruginosa* ; *A. baumannii*: *Acinetobacter baumannii*

4. Discussion

A total of 81 bacterial strains, including *Staphylococcus aureus*, *Pseudomonas aeruginosa*, coagulase-negative staphylococci, and *Acinetobacter baumannii*, were isolated from the urine of patients with urinary catheters. Of these strains, *Staphylococcus aureus* was predominant, with an isolation rate of 44.44%. This bacterial profile is consistent with several studies conducted in hospital settings, which report a strong involvement of *Staphylococcus aureus* and *Pseudomonas aeruginosa* in catheter-associated urinary tract infections, as well as the notable presence of coagulase-negative staphylococci, often underestimated but relevant in this type of infection [10].

The isolation of these strains highlights the bacterial diversity involved in urinary device-associated infections. The predominance of *Staphylococcus aureus* in this study is a significant finding. This bacterium is well known for its ability to colonize medical devices and form biofilms, which promotes infection persistence and resistance to antimicrobial treatments [11].

The presence of *Pseudomonas aeruginosa*, coagulase-negative staphylococci, and *Acinetobacter baumannii* also underscores the complexity of catheter-related urinary tract infections, as these microorganisms are frequently implicated in nosocomial infections. *Pseudomonas aeruginosa* is particularly recognized for its multiple

mechanisms of antibiotic resistance and its ability to survive in hospital environments, according to Poole [12]. Similarly, *Acinetobacter baumannii* is an emerging opportunistic pathogen, often associated with multidrug-resistant infections, particularly in immunocompromised patients or those with medical devices [13]. However, one study highlighted that enterobacteria, particularly *Escherichia coli* and *Klebsiella pneumoniae*, were the major etiological agents of urinary tract infections in these patients [14].

The analysis revealed a predominance of infection in men (85%) compared to 15% in women. This prevalence corroborates that of Sanou et al. [14], who, in their study of 47 patients with catheters, found that 44 were male, representing 93.6% of the sample. This predominance could be explained by several factors such as age, comorbidities, and care practices. For example, in men over 50, the incidence of urinary tract infections increases due to prostatic hypertrophy and the use of catheters, which may explain a higher prevalence in this population [15].

The results indicated that the coagulase-negative *Staphylococcus* (CNS) strains tested were completely resistant (100%) to cefoxitin. This rate is higher than that reported by Martins et al. [16]. These authors reported that 47% of CNS isolates were resistant to cefoxitin, with a prevalence of 47% methicillin resistance. The total resistance of these strains observed in this study is a strong indicator of the likely presence of resistance mechanisms mediated by the *mecA* gene, characteristic of methicillin-resistant *Staphylococcus* strains [17]. Furthermore, according to Becker et al. [18], high resistance to beta-lactams complicates clinical management and antibiotic therapy.

Acinetobacter baumannii isolates exhibited high resistance rates (100%) to almost all of the beta-lactams tested. This result differs from that obtained in a study conducted at the Mohammed V Military Teaching Hospital in Rabat, Morocco. It reported a high resistance to ticarcillin of 72.3% [19].

Analysis of the results obtained showed that the strains of *Acinetobacter baumannii* and *Pseudomonas aeruginosa* expressed resistance rates of 100% and 50%, respectively, to ciprofloxacin. Several scientific studies have observed high resistance rates of these bacteria to ciprofloxacin, although the percentages vary according to geographical contexts and the periods studied. Indeed, a retrospective study conducted over seven years in a burn intensive care unit reported that the resistance of *Pseudomonas aeruginosa* to ciprofloxacin was 42.9% in 2018, while that of *Acinetobacter baumannii* was 90.5% during the same period [20]. In Tunisia, Mellouli et al. [21] found that *Acinetobacter baumannii* resistance to ciprofloxacin was 93.5%, with colistin resistance of 1.8%. These variations can be attributed to factors such as local antibiotic use practices, infection control protocols, and differences in data collection and analysis methods.

Regarding the detection of *qnr* genes in this study, analysis of the results shows that resistance genes were present at varying rates. Specifically, *qnr* genes B and S were predominant, with rates of 80% and 62.5%, respectively, in 32 and 25 strains. *qnr* A was present in 5 strains at a rate of 12.5%. The variable presence of *qnr* resistance genes in the studied strains reflects the diversity

of genetic mechanisms involved in quinolone resistance, particularly to ciprofloxacin. The *qnr* genes encode proteins that protect DNA gyrase and topoisomerase IV, the main targets of quinolones, thus reducing the efficacy of these antibiotics [22].

In this study, the predominance of the *qnrB* (80%) and *qnrS* (62.5%) genes is consistent with several reports that have also identified these genes as the most frequent in clinical strains of *Pseudomonas aeruginosa* and *Acinetobacter baumannii*. For example, a systematic review published by Cattoir and Nordmann [23] highlights that the *qnrB* and *qnrS* genes are often associated with high quinolone resistance in Enterobacteriaceae and non-fermenting bacteria. The lower presence of the *qnrA* gene (12.5%) confirms that this variant is less frequent in these species, although it is also an important factor in plasmid-mediated quinolone resistance [24]. This variable distribution of *qnr* genes underscores the importance of molecular surveillance for better understanding region-specific resistance profiles and adapting therapeutic strategies.

The results showed that *Acinetobacter baumannii* strains expressed different types of co-expression: *qnr* A-B, *qnr* B-S, and *qnr* A-B-S, with levels of 50%, 100%, and 50%, respectively. The results indicating co-expression of *qnr* A-B, *qnr* B-S, and *qnr* A-B-S genes in *Acinetobacter baumannii* strains demonstrate the complexity and multiplicity of the genetic mechanisms responsible for quinolone resistance. The co-presence of several *qnr* genes in the same strain can amplify protection against quinolones by providing more robust resistance that is potentially more difficult to overcome by antibiotic treatments [22]. The high co-expression rate of the *qnr* B-S type (100%) suggests that this combination could be particularly advantageous for bacterial survival under antibiotic pressure, which is consistent with previous observations where the *qnrB* and *qnrS* genes are frequently detected together in mobile plasmids, thus facilitating their dissemination between bacteria [23].

Although less frequent, *qnr* A-B (50%) and *qnr* A-B-S (50%) co-expressions indicate significant genetic diversity that could contribute to varied resistance profiles. Such diversity can complicate the clinical management of infections, as it can lead to different levels of resistance and response to treatments [24].

5. Conclusion

The presence of *qnr* genes detected in the pathogens in this study suggests the susceptibility of these genes to dissemination via horizontal gene transfer mechanisms. The *Acinetobacter baumannii* strains expressed different types of co-expression. The resistance profile showed that coagulase-negative *Staphylococcus* and *Acinetobacter baumannii* strains expressed 100% resistance in the presence of cefoxitin, respectively. However, amikacin showed greater activity against *Pseudomonas aeruginosa* (n=28) and *Acinetobacter baumannii* strains. The information on antibiotic resistance obtained in this study highlights the need for rigorous antibiotic management to prevent the spread of resistant strains.

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Authors Contribution

Eric Joel Tahou and Kouadio Fernique Konan carried out sample collection and processing, data collection, laboratory analyses, and interpretation. Bertin Tiecoura performed data analysis, statistical analyses, results visualization, and literature reviews. Assanvo Simon-Pierre N'guetta and Kouadio Nathalie Guesseend provided critical feedback on the manuscript and supervised this study. All authors contributed to the drafting, review, and approval of the final version of the manuscript.

Competing Interests

The authors have no competing interests.

References

- [1] World Health Organization (WHO), Antimicrobial resistance. Fact sheets. Disponible sur: <https://www.who.int/news-room/fact-sheets/detail/antimicrobial-resistance> 2020.
- [2] Davies, J. and Davies, D, Origins and evolution of antibiotic resistance. *Microbiology and Mol. Biol Rev.* 74(3):417-433 Septembre 2010.
- [3] Lardi D, Impact of efflux in fluoroquinolone and ethium bromide resistance in *Clostridium difficile*. Thèse de doctorat, Université Paris 11. Disponible sur: <https://theses.fr/2005PA114822>, 2005.
- [4] Ventola, C.L, The antibiotic resistance crisis: part 1: causes and threats. *P T.* 40(4):277-283, Avril 2015.
- [5] Public Health France, National Surveillance of Fluoroquinolone Resistance in Urinary *Escherichia coli* Isolates 2015-2019. 2020. Available at: <https://www.santepubliquefrance.fr/maladies-et-traumatismes/infections-associees-aux-soins-et-resistance-aux-antibiotiques/resistance-aux-antibiotiques/documents/article/surveillance-nationale-de-la-resistance-aux-cephalosporines-de-3e-generation-et-aux-fluoroquinolones-des-isolats-urinaires-d-escherichia-coli>.
- [6] Nsofor, C.M., Tattfeng, M.Y. and Nsofor, C.A, High prevalence of *qnrA* and *qnrB* genes among fluoroquinolone-resistant *Escherichia coli* isolates from a tertiary hospital in southern Nigeria. *Bull Natl Res Cent* 45, 26. Décembre 2021.
- [7] Mabika, M.R. Mounioko, F., Souza, A. and Yala, J.F, Phenotypic and molecular characterization of quinolone resistance in enteropathogens isolated from diarrhea in young children in Koula-Moutou, Gabon. *Egypt. J. Basic Appl. Sci.* 8 (1), 345–353. Novembre 2021.
- [8] Kouakou, M.F., Toty, A.A., Gadou, V., M'Bengue, G.V., Konan, K.F., Guédé, K.B., Ouattara, M.B., Tiécoura, K., Guesseend, N. and Dosso, M, Detection of *qnr* Genes That Mediate Fluoroquinolone Resistance in Gram-Negative Bacilli in Abidjan, Côte d'Ivoire. *Am. J. Microbiol. Res.* 11(3), 79-82. October 2023.
- [9] EUCAST-CASFM: Antibigram Committee of the French Society of Microbiology (2023). https://www.sfm-microbiologie.org/wp-content/uploads/2023/06/CASFM2023_V1.0.pdf.
- [10] Genao, L. and Buhr, G.T, Urinary Tract Infections in Older Adults Residing in Long-Term Care Facilities. *Ann Longterm Care*; 20(4):33-38. April 2012.
- [11] Otto, M, *Staphylococcus aureus* toxins. *Curr Opin Microbiol.* 17:32-7. February 2014.
- [12] Poole, K, *Pseudomonas aeruginosa*: resistance to the max. *Front Microbiol.* 2:65. April 2011.
- [13] Peleg, A.Y., Seifert, H. and Paterson D.L, *Acinetobacter baumannii*: emergence of a successful pathogen. *Clin Microbiol Rev.* 21(3):538-582. July 2008.
- [14] Sanou, A., Gnada, K., Traore, I. and Sanou, S, Frequency and microbiological profiles of urinary tract infections in patients with indwelling catheters at the Bacteriology Laboratory of the Muraz Center. *Science and Technology, Health Sci.*, 44(2), 72–79. April 2024.
- [15] Foxman, B, Urinary tract infection syndromes: occurrence, recurrence, bacteriology, risk factors, and disease burden. *Infect Dis Clin North Am.* 28(1): 1-13. Mars 2024.
- [16] Martins, M.A., Santos, S., Leão L.S., Araújo, N.P. and Bacion, M.M, Prevalence of resistance phenotypes in *Staphylococcus aureus* and coagulase-negative isolates from venous ulcers in primary care patients. *Rev Soc Bras Med Trop*; 45(6): 717-22. December 2012.
- [17] Kahl, B.C., Becker, K. and Löffler, B, Clinical Significance and Pathogenesis of *Staphylococci* in the Human Microbiome. *Clin Microbiol Rev.*; 29(2):401-27. April 2016.
- [18] Becker, K., Heilmann C. and Peters, G, (2014). coagulase-negative *Staphylococci*. *Clin Microbiol Rev.*, 27(4):870-926. October 2014.
- [19] Elouennass, M., Bajou, T., Lemnouer, A.H., Foissaud, V.H.V. and Baaj, A.J, *Acinetobacter baumannii*: Study of the susceptibility of strains isolated at the Mohammed V Military Teaching Hospital, Rabat, Morocco. *Med Infect Dis*, (33) 7, P. 361-364. July 2003.
- [20] Krir, A. Dhraief, S. Messadi, A. A. and Thabet, L, Bacteriological profile and antibiotic resistance of bacteria isolated in a burn intensive care unit over seven years. *Ann Burns Fire Disasters*, 32(3):197-202. September 2019.
- [21] Mellouli, A., Maamar, B., Bouzakoura, F., Messadi, A.A. and Thabet, L, Colonization and infection with *Acinetobacter baumannii* in a burn intensive care unit in Tunisia. *Ann Burns Fire Disasters*. 34(3):218-225. Septembre 2021.
- [22] Robicsek, A., Jacoby, G.A. and Hooper, D.C, The worldwide emergence of plasmid-mediated quinolone resistance. *Lancet Infect Dis.*, 6(10), 629-640. October 2006.
- [23] Ezech, P. A., Olayinka, B. O. and Bolaji, R.O, Plasmid Mediated Quinolone Resistance in *Enterobacteriaceae*: A Review, *NJM*, 34(1):-4962-4976.
- [24] Poirel, L., Cattoir, V and Nordmann, P, Plasmid-Mediated Quinolone Resistance; Interactions between Human, Animal, and Environmental Ecologies. *Front Microbiol.* 2; 3: 24. February 2012.



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