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Research Article

Characterization of Quinolone Resistance Genes in Gram-Negative Bacilli Isolated from Pus and Vaginal Swabs at Saint Camille Hospital, Ouagadougou (HOSCO), Burkina Faso

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Abstract

Introduction: Antibiotic resistance, particularly to quinolones, represents a major public health concern in Burkina Faso. In recent decades, plasmid-mediated quinolone resistance (PMQR) mechanisms have emerged, especially among Gram-negative bacteria. These mechanisms include, among others, *qnr* genes (*qnrA*, *qnrB*, *qnrS*). This study aimed to investigate the presence of quinolone resistance determinants in Gram-negative bacilli isolated from pus and vaginal samples at Saint Camille Hospital of Ouagadougou (HOSCO). *Methodology:* A total of 19 strains of *Escherichia coli* isolated from pus and vaginal swabs were collected for bacteriological and molecular analysis. Four antibiotics, namely ciprofloxacin (CIP), norfloxacin (NOR), ofloxacin (OF) and levofloxacin (LEV) were used for sensitivity testing and molecular analysis focused on the detection of qnrA, qnrB, and qnrS type genes. *Results:* Resistance rates to CIP, NOR, OF, and LEV were 57.89%, 52.63%, 52.63%, and 31.37%, respectively. Molecular analysis revealed the presence of *qnrB*, and *qnrS* genes in 28.47% of the isolates, for each gene. The *qnrA* gene was not detected in any isolate. The analysis of the genetic support of resistance genes revealed that 50% of the *qnrS* genes were plasmid-borne, while only 25% of the *qnrB* genes were associated with plasmids. La Correlation analysis between resistance genes and antibiotics showed a moderate positive correlation between *qnrB* and NOR/LEV, thereby suggesting the involvement of *qnrB* in resistance to these antibiotics. *Conclusion:* These findings highlight the need for continuous surveillance of antibiotic resistance in clinical isolates.

Keywords

Quinolones, *Qnr Genes*, Burkina Faso

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1. Introduction

Quinolones are a class of broad-spectrum synthetic antimicrobial agents commonly used to treat infections caused by Gram-negative bacteria. Their primary mechanism of action involves the inhibition of DNA gyrase and topoisomerase IV, enzymes essential for bacterial DNA replication and transcription [1-9]. For many years, quinolone resistance was believed to arise exclusively through chromosomal mechanisms—particularly mutations in genes encoding DNA gyrase and topoisomerase IV, overexpression of efflux pumps leading to active drug extrusion, and reduced outer membrane permeability due to porin loss [10-14].

However, over the past few decades, plasmid-mediated quinolone resistance (PMQR) mechanisms have emerged, particularly among Gram-negative organisms [15]. These mechanisms include the *qnr* genes (*qnrA*, *qnrB*, *qnrC*, *qnrD*, *qnrS*, and *qnrVC*), which encode proteins that protect DNA gyrase from quinolone binding [1, 15, 16]; enzymes such as the aminoglycoside acetyltransferase encoded by *aac(6')-Ib-cr*, which modifies certain fluoroquinolones [1, 10, 15, 17]; and efflux pump genes such as *qepA* and *oqxAB*, which actively expel quinolones from the bacterial cell [8, 18, 19].

Although *qnr* genes alone typically confer low-level resistance, their presence facilitates the selection of additional chromosomal mutations that can lead to high-level resistance, thereby contributing to the alarming global rise in quinolone resistance [2, 20].

In Burkina Faso, data on the distribution of quinolone resistance genes remain scarce. This study aims to contribute to the understanding of resistance profiles among Gram-negative bacilli isolated from clinical samples. Specifically, we seek to detect the presence of key plasmid-mediated quinolone resistance determinants (*qnrA*, *qnrB* and *qnrS* in isolates obtained from pus and vaginal specimens collected at Saint Camille Hospital of Ouagadougou (HOSCO).

2. Methodology

2.1. Sample Collection

A total of 19 clinical samples—including 14 pus samples and 5 vaginal swabs—were collected from patients at Saint Camille Hospital of Ouagadougou (HOSCO) over a one-month period, from March to April 2024.

2.2. Bacteriological Analysis

Samples were subjected to standard bacteriological procedures, including macroscopic examination, inoculation and incubation, microscopic observation, bacterial isolation, identification, and antibiotic susceptibility testing. Bacterial isolation was performed on Eosin Methylene Blue selective agar. Once isolated, bacterial strains were identified using biochemical assays, specifically the API 20E system, according to the manufacturer's instructions.

Antimicrobial susceptibility testing was conducted using the disc diffusion method on Mueller-Hinton agar, in accordance with the guidelines of the French Society for Microbiology's Antibiogram Committee (CASFM). The antibiotics tested included ciprofloxacin (CIP), norfloxacin (NOR), levofloxacin (LEV), and ofloxacin (OF).

2.3. Bacterial DNA Extraction

Genomic DNA was extracted from the isolated bacterial strains using the boiling method. Bacterial cells were lysed by heating at 100 ℃ to release their genetic material. The lysate was then centrifuged to remove cellular debris, and the supernatant containing DNA was collected. DNA concentration and purity were measured using a Nanodrop spectrophotometer prior to PCR amplification.

2.4. Polymerase Chain Reaction (PCR)

Table 1. *Primer sequences used for the detection of quinolone resistance genes.*

Genes	Primer Sequences (5' → 3')	Amplicon Size (bp)	References
qnrA	For: ATTTCTCACGCCAGGATTTG Rev: GATCGGCAAAGGTTAGGTCA	516 bp	[21]
qnrB	For: GATCGTGAAAGCCAGAAAGG Rev.: ACGATGCCTGGTAGTTGTCC	469 bp	[21]
qnrS	For: ACGACATTCGTCAACTGCAA Rev: TAAATTGGCACCCTGTAGGC	417 bp	[21]

Conventional PCR was employed to detect the presence of quinolone resistance genes (*qnrA*, *qnrB* and *qnrS*, using gene-specific primers (see Table 1). Amplified products were subsequently visualized via agarose gel electrophoresis.

2.5. Plasmid DNA Extraction

In order to determine the genetic support of the identified resistance genes, plasmid DNA extraction was performed on the strains harboring at least one of the targeted genes. The alkaline lysis method was employed for this purpose. Following plasmid quantification, conventional PCR was carried out using the primers previously described (Table 1).

2.6. Data Analysis

Data were compiled using Microsoft Excel and statistically analyzed with R software, version 4.3.3.

3. Results

Antibiotic susceptibility testing was conducted for four fluoroquinolones: ciprofloxacin (CIP), levofloxacin (LEV), ofloxacin (OF), and norfloxacin (NOR). The presence of quinolone resistance genes—*qnrA*, *qnrB* and *qnrS*, was also assessed. Molecular analysis was performed on bacterial isolates exhibiting resistance to at least one of the tested antibiotics.

3.1. Distribution of Bacterial Species

The analysis of bacterial species distribution (Table 2) revealed that *Escherichia coli* was the most prevalent species, identified in 10 isolates (7 from pus samples and 3 from vaginal swabs). *Klebsiella pneumoniae* was detected in 5 isolates (3 pus, 2 vaginal), followed by *Pseudomonas aeruginosa* in 3 isolates (all from pus), and *Proteus mirabilis* in a single isolate (pus).

Table 2. *Distribution of Bacterial Species.*

Species	Pus samples	Vaginal swabs
E. coli	7	3
K. pneumoniae	3	2
P. aeruginosa	3	0
P. mirabilis	1	0

3.2. Antibiotic Resistance Profile

The results of the antibiotic susceptibility testing (Table 3) revealed considerable variability in resistance rates depending on the antibiotic tested.

Table 3. *Antibiotic Susceptibility Profile.*

Identification Number	Sex	Bacterial Species	CIP	LEV	OF	NOR
Pus 2	M	*K. pneumoniae*	S	S	R	S
Pus 11	F	*P. aeruginosa*	R	S	S	R
Pus 10	M	*E. coli*	R	R	R	R
Pus 1	M	*E. coli*	R	R	R	S
PV32	F	*K. pneumoniae*	R	S	R	R
PUS 5931	F	*E. coli*	R	R	R	R
PUS42	F	*E. coli*	R	R	R	R
PV2	M	*E. coli*	R	S	S	R
Pus 9	M	*P. mirabilis*	S	R	R	S
Pus 8	F	*K. pneumoniae*	R	R	R	R
Pus 5	F	*E. coli*	S	S	S	S
Pus 4	M	*P. aeruginosa*	R	S	S	R
Pus 3	M	*E. coli*	R	S	R	R
Pus 12	F	*E. coli*	R	S	R	R
PV32	F	*E. coli*	S	S	S	S
PV12	F	*K. pneumoniae*	S	S	S	S

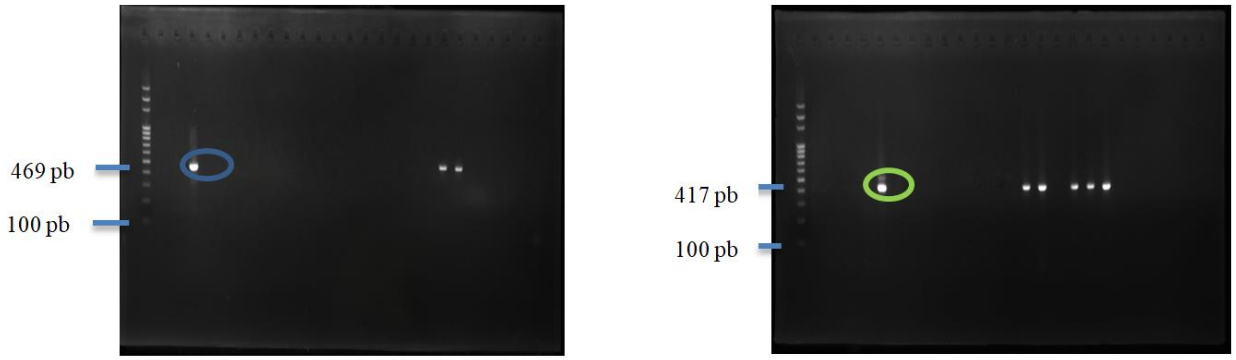
Identification Number	Sex	Bacterial Species	CIP	LEV	OF	NOR
PV 1	M	*E. coli*	S	S	S	S
Pus 7	M	*K. pneumoniae*	S	S	S	S
Pus 6	F	*P. aeruginosa*	S	S	S	S

This table demonstrates a high level of resistance to ciprofloxacin (CIP), with 11 resistant isolates (57.9%), followed by ofloxacin (OF) and norfloxacin (NOR), each exhibiting resistance in 10 isolates (52.63%). Levofloxacin (LEV) showed a moderate resistance level, with 6 resistant isolates (31.6%).

Table 4. *Distribution of Resistance Genes.*

Genes	Presence/Absence	Frequency	Total	Percentage
qnrA	Négative	14	14	100%
qnrB	Négative	10	14	71.4%
qnrB	Positive	4	14	28.6%
qnrS	Négative	10	14	71.4%
qnrS	Positive	4	14	28.6%

The table above reports, for each resistance gene (*qnrA*, *qnrB*, *qnrS*), the number of bacterial isolates identified as either "Positive" or "Negative." The *qnrB* and *qnrS* genes exhibited the same distribution pattern, each being present in 4 isolates (28.6%) and absent in 10. In contrast, the *qnrA* gene was not detected in any of the tested isolates. The PCR results, as illustrated by the images obtained, are shown in Figure 1.



Electrophoretic profile of PCR products targeting qnrB gene on agarose gel

Electrophoretic profile of PCR products targeting qnrS gene on agarose gel

Figure 1. *Visualization of PCR Amplification Products by Agarose Gel Electrophoresis.*

3.3. Genetic Support of Resistance Genes

The test for determining the genetic support of resistance genes yielded the following results:

Table 5. *Genetic Support Assessment of Antimicrobial Resistance Genes.*

Genes	Positive	Negative
P-qnrS	2 (50%)	2 (50%)
p-qnrB	1 (25%)	3 (75%)

As shown in Table 5 above, among the four qnrS-type genes identified, 50% were carried by plasmids. In contrast, only 25% of the qnrB-type genes were found to be plasmid-borne.

3.4. Correlation Between Resistance Genes and Antibiotic Susceptibility

The correlation analysis between resistance genes and antibiotic susceptibility profiles (Figure 2) revealed a significant positive correlation between *qnrB* and NOR (r = 0.40), as well as with LEV (r = 0.33). Conversely, a strong negative correlation was observed between *qnrS* and OF (r = –0.548).

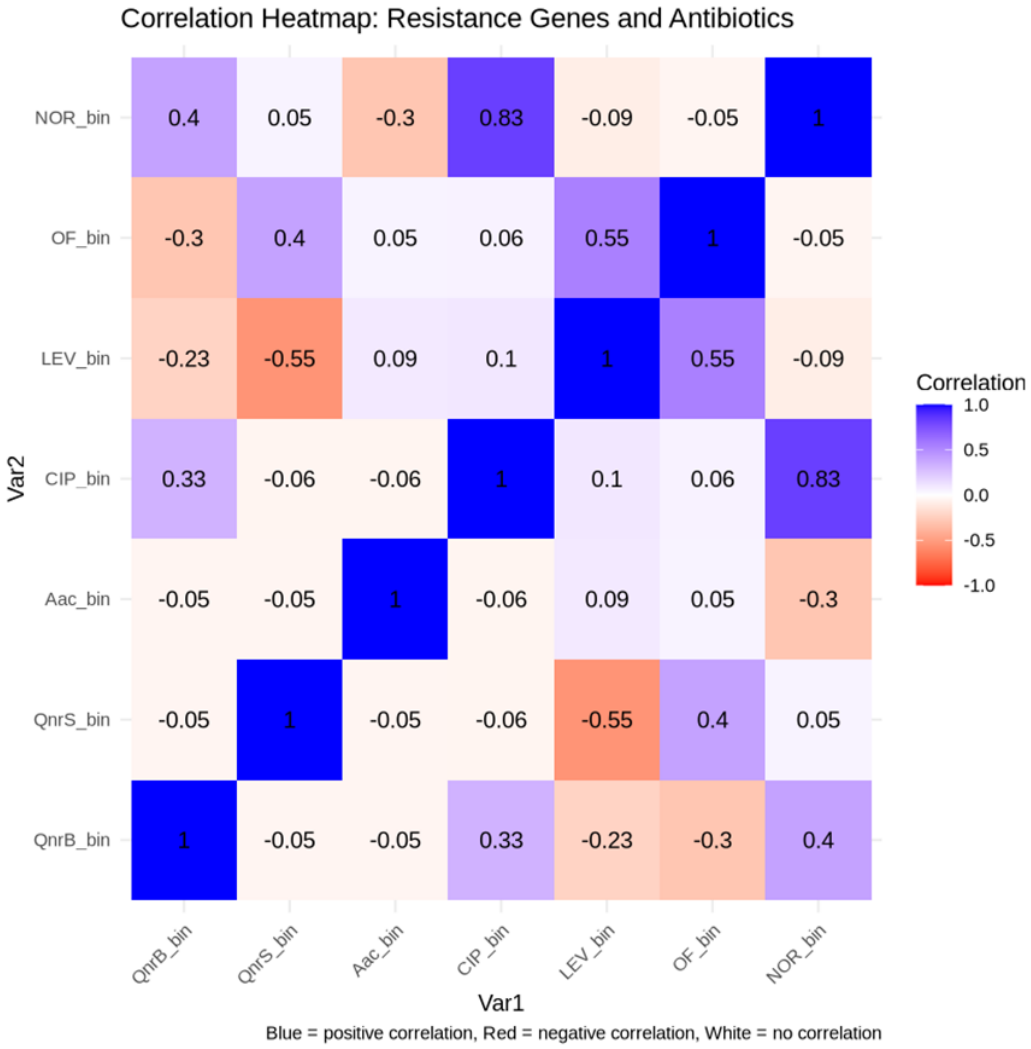


Figure 2. *Heatmap of Correlations Between Resistance Genes and Antibiotics.*

The heatmap illustrates strong positive correlations (in blue), strong negative correlations (in red), and weak or negligible correlations (in white).

4. Discussion

The emergence of antibiotic-resistant bacteria, particularly those resistant to quinolones, poses a serious threat to the effectiveness of antimicrobial agents that have revolutionized medicine and saved millions of lives [22, 23]. This challenge is particularly alarming in low-resource countries such as Burkina Faso, where infectious diseases, poverty, and malnutrition remain endemic [24]. Prompt administration of appropriate antimicrobial therapy is essential for effective infection management. Empirical treatment strategies must therefore be informed by a thorough understanding of the pathogens involved and their resistance profiles [25].

The aim of this study was to assess the prevalence of fluoroquinolone resistance among Gram-negative bacilli and to identify the associated resistance genes. Among the isolates analyzed, *E. coli* was the most frequently detected species (52.63%), a finding consistent with several previous studies [26-31]. However, other reports have identified *K. pneumoniae* as the dominant species [32, 33].

In the present study, high resistance rates were observed for ciprofloxacin (CIP, 57.89%), norfloxacin (NOR, 52.63%), and ofloxacin (OF, 52.63%), with levofloxacin (LEV) exhibiting a comparatively lower resistance rate (31.37%). Similar trends have been reported elsewhere—for example, Swedan

et al. found ciprofloxacin resistance to be predominant in Jordan [1], while Rafya *et al.* reported a high resistance rate to levofloxacin (93.4%) in Iraq [22]. Differences across studies may be attributed to various factors, including population socio-economic characteristics, methodological differences, antibiotic accessibility, patterns of antibiotic misuse, and the clinical origin of the isolates.

Among the known mechanisms of fluoroquinolone resistance, plasmid-mediated resistance is one of the most recently identified [17]. This study specifically investigated three *qnr* genes (*qnrA*, *qnrB*, *qnrS*). The *qnrB* and *qnrS* genes were each detected in 28.57% of the isolates, while *qnrA* was not detected in any sample. These frequencies are lower than those reported in a study from Togo, where the prevalence of *qnrB*, *qnrS*, and *qnrA* was 47.74%, 47.10%, and 2.58%, respectively [5]. In studies conducted in Morocco [31], Mexico [34], and Côte d'Ivoire [13], *qnrB* was the most commonly identified gene.

In our dataset, *qnr* gene prevalence was highest in *E. coli* isolates (28.57%). This contrasts with studies from Tunisia and South Korea, where *K. pneumoniae* exhibited a significantly higher prevalence of *qnr* genes (up to 80%) [19, 35]. Such discrepancies may reflect regional variation in circulating resistance genes and phenotypes, as well as differences in sample size and bacterial strain distribution. The analysis of the genetic support of resistance genes revealed that 50% of the *qnrS* genes were plasmid-borne, while only 25% of the *qnrB* genes were associated with plasmids. However, several studies have reported and confirmed that *qnr* genes are typically carried by plasmids [1, 15, 16]. This discrepancy could be explained by the possibility that the bacterial strain under investigation may have lost the plasmid carrying the *qnr* gene during culture or isolation. Additionally, the *qnr* gene may be located on other mobile genetic elements, such as transposons or integrons, which are not necessarily plasmid-associated but can still facilitate chromosomal integration. It is also possible that *qnr* genes are directly integrated into the bacterial chromosome rather than being carried by mobile genetic elements such as plasmids.

We also conducted a correlation analysis to explore potential relationships between resistance genes and the antibiotics to which they confer resistance. A significant positive correlation was found between *qnrB* and both NOR (r = 0.40) and LEV (r = 0.33). Conversely, *qnrS* showed a strong negative correlation with OF. The observed positive correlation supports the role of *qnrB* in resistance to NOR and LEV. Meanwhile, the negative correlation suggests the presence of alternative resistance mechanisms, such as chromosomal mutations in genes encoding DNA gyrase or overexpression of efflux pumps [4, 36].

5. Conclusion

This study investigated quinolone resistance in various Gram-negative bacilli isolated from pus and vaginal swab samples. A high prevalence of resistance to ciprofloxacin, norfloxacin, and ofloxacin was observed. Additionally, a significant presence of specific resistance genes (*qnrB*, *qnrS*) was noted. These findings underscore the importance of continuous monitoring of antibiotic resistance in clinical isolates to inform effective antimicrobial stewardship.

Abbreviations

HOSCO	Saint Camille Hospital of Ouagadougou
PMQR	Plasmid-Mediated Quinolone Resistance
CIP	Ciprofloxacin
NOR	Norfloxacin
LEV	Levofloxacin
OF	Ofloxacin
DNA	Deoxyribonucleic Acid
EMB	Eosin Methylene Blue
CASFM	French Society for Microbiology's Antibiogram Committee

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Conflicts of Interest

The authors declare no conflicts of interest.

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