microorganisms

MDPI

Article

Determination of Resistance Profiles to Fluoroquinolones and Characterization of Genetic Determinants of Resistance in *Campylobacter* spp. Isolated from Diarrheal Faeces in Children in the City of Makokou

Jean Fabrice Yala [1,2,*], Rolande Mabika Mabika [2], Ornella Zong Minko [1,2], Evrard Mayombo Ngoussou [2], Léonce Fauster Ondjiangui [2], José-Miguel Armesto Paula [3], Junior Aymard Ondo-Nang [3] and Franck Mounioko [4]

[1] Agrobiology Research Unit, Molecular and Cellular Biology Laboratory, Microbiology Team (LABMC), Masuku University of Sciences and Techniques (USTM), Franceville BP 067, Gabon; ornellapacy@gmail.com
[2] Bacteriology Laboratory, Medical Analysis Research Unit, Interdisciplinary Center for Medical Research of Franceville (CIRMF), Franceville BP 769, Gabon; rolandemabika@gmail.com (R.M.M.); mayombo.n.evrard@gmail.com (E.M.N.); leoncefauster@gmail.com (L.F.O.)
[3] Centre Hospitalier Régional Omar Bongo ONDIMBA de Makokou (CHROBOM), Makokou, Gabon; armestopaulajosemiguel@gmail.com (J.-M.A.P.); ondojunior266@gmail.com (J.A.O.-N.)
[4] Vector Systems Ecology Research Unit, Interdisciplinary Center for Medical Research of Franceville (CIRMF), Franceville BP 769, Gabon; fmounioko@yahoo.fr
* Correspondence: yalajeanfabrice@gmail.com; Tel.: +241-66-18-84-88

Abstract

Campylobacter is a zoonotic bacterium and the leading cause of bacterial gastroenteritis in humans and animals in several countries. It is managed through antibiotic therapy, including macrolides (erythromycin) and fluoroquinolones (ciprofloxacin), which is the drug of choice. The aim of this study was to investigate the phenotypic resistance profile and the molecular determinants potentially associated with fluoroquinolone resistance in *Campylobacter* isolates obtained from diarrhoeal faeces of children under 5 years of age in Makokou, Gabon. The susceptibility of 116 *Campylobacter* isolates was evaluated against ciprofloxacin and pefloxacin (second-generation fluoroquinolones) and levofloxacin (third-generation fluoroquinolone). The genetic basis of any resistance to these compounds was determined by screening the chromosomal *gyrA* gene and the plasmid *qnrA, qnrB* and *qnrS* genes, which were detected using conventional PCR. Phenotypically, all isolates are resistant to all compounds (100%). Molecularly, the *gyrA* gene was amplified in 94.0% of the isolates, the *qnrS* gene in 51.7% (60/116), the *qnrB* gene in 4.3% (5/116), and the *qnrA* gene 0.0%. Furthermore, 52.6% (61/116) carried both the *gyrA* gene and at least one of the *qnr* genes detected. *Campylobacter* isolates showed total resistance to fluoroquinolones, suggesting a probable therapeutic deadlock with these molecules, possibly mediated by *qnr* genes. The detection of these genetic determinants suggests that plasmid-mediated transmission may contribute to the spread of resistant *Campylobacter* in various environments. However, given that conventional PCR only confirms the presence of the *gyrA* gene and not the specific mutations within the quinolone resistance-determining region (QRDR) that confer resistance, additional genomic sequencing is required to fully elucidate the mechanisms of resistance. This study consequently highlights the importance of monitoring the epidemiological evolution and resistance of *Campylobacter* isolates in order to optimize therapeutic management strategies.

Check for updates

Academic Editor: Andreas E. Zautner

Received: 16 October 2026
Revised: 10 November 2026
Accepted: 15 November 2026
Published: 18 November 2026

Copyright: © 2026 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license.

Keywords: fluoroquinolone; *qnr*; *gyrA*; *Campylobacter*; Gabon

1. Introduction

Campylobacter is a Gram-negative, spiral-shaped, microaerophilic bacterium that can take on a coccoid form under unfavourable conditions, such as in the presence of excess oxygen. *Campylobacter* is a zoonotic pathogen prevalent in the intestinal tract of wild and domestic animals. It is the leading cause of gastroenteritis in humans and animals in many countries and of foodborne illnesses [1–3]. In fact, in sub-Saharan Africa, the prevalence of *Campylobacter* was estimated to be between 1.7% and 62.7% in humans and between 1.2% and 80% in animals [4]. Furthermore, the spread of this pathogen is facilitated by factors such as agriculture, livestock farming, water quality, and sanitation facilities [5]. *Campylobacter* is responsible for several infections characterized by watery or bloody diarrhea, abdominal pain, fever, and nausea [6]. However, the incidence of *Campylobacter* infections has been rising steadily in recent years. Indeed, in 2024, the European Centre for Disease Prevention and Control (ECDC) confirmed 140,241 cases of *Campylobacter* infection in Europe [2].

Globally, broiler chicken production has increased approximately sevenfold since the 1960s, often involving high-density, caged rearing. Chickens are the primary reservoir for *Campylobacter*; furthermore, high-density group rearing greatly facilitates horizontal transmission within flocks, the exchange of genes between strains, and the emergence of highly transmissible, poultry-adapted strains [6]. Cross-contamination during slaughter and processing is very common. Moreover, international tourism and the import and export of meat products via the cold chain facilitate the cross-border spread of antimicrobial-resistant strains. Combined with the use of veterinary antibiotics, which drive the selection and persistent dissemination of resistant strains, these factors perpetuate the cycle of resistance and complicate therapeutic management in humans [7,8].

Campylobacter infections are treated with appropriate antibiotic therapy, which includes macrolides with erythromycin as the first-line agent as well as fluoroquinolones (FQs), such as ciprofloxacin, which serve as a second-line alternative to be considered if the first-line treatment proves ineffective [1,3]. However, the inappropriate, indiscriminate, and abusive use in both human and veterinary medicine has generated and increased the growth of resistance to FQs, to the point that the World Health Organization (WHO) classifies FQ-resistant *Campylobacter* as high-priority pathogens for the development of new antibiotics [1,3]. Indeed, several studies have highlighted the resurgence of the increase in strains resistant to ciprofloxacin and other molecules [1,3]. Notably, a study conducted in Rwanda on *Campylobacter* isolates from patients attending clinical facilities in Kigali showed a ciprofloxacin resistance rate of 37.1%, as well as high resistance rates to streptomycin (82.5%) and erythromycin (66.7%) [3]. In Kenya, the resistance rates are 87.5% for erythromycin, 75.0% for doxycycline, 73.7% for ampicillin, 73.3% for cotrimoxazole, and 68.8% for minocycline [7].

However, several studies have shown that resistance to FQs is mainly the result of chromosomal mutations, particularly in DNA gyrase. The C257T nucleotide substitution, resulting in the Thr86Ile (T86I) amino acid substitution, is the most frequently reported alteration associated with high-level FQ resistance [4–6]. However, it has also been demonstrated in the literature that plasmid-mediated resistance due to the Qnr complex constitutes the second mechanism of resistance and that this emergence has been increasing over time. Finally, in addition to these two mechanisms, the CmeABC efflux system, an efflux pump that plays an essential role in transporting substances out of the bacterial cell, confers high-level resistance to several classes of antibiotics [8]. Moreover, recent work has shown that among *Campylobacter* isolates, multidrug-resistant (MDR), extensively drug-resistant

(XDR) and pandrug-resistant (PDR) isolates comprise 75%, 20.8% and 4.2%, respectively, which exacerbates this crisis [9].

In response to the increase in levels of resistance to FQs and other antimicrobial molecules, restrictions on the use of FQs in livestock have been implemented in the European Union (2003 and 2006) and in the United States (2005), as well as surveillance and monitoring systems for antimicrobial resistance [1].

In Gabon, there is a significant lack of health systems for monitoring and tracking the epidemiology of *Campylobacter* bacteria, their antibiotic resistance, as well as empirical prescription, sale, and inappropriate and abusive use of antibiotics. FQs are the first-line molecules prescribed for the treatment of urinary and diarrhoeal infections in healthcare facilities in Gabon. So, in this national context of the absence of data on *Campylobacter*, it seems appropriate to know their antibiotic resistance profiles. Therefore, this study's main objectives are to determine the resistance profile of *Campylobacter* spp. isolates from diarrhoeal faeces of children under 5 years old in the city of Makokou to fluoroquinolones and to characterize the genetic determinants of resistance to these molecules. Specifically, it will involve determining their sensitivity to second-, third-, and fourth-generation FQs; searching for plasmid genes of the *qnr* complex; and searching for the wild-type chromosomal gene *gyrA*.

2. Materials and Methods

2.1. Bacterial Strains

The different *Campylobacter* isolates used in this study were obtained from the strain collection of the Interdisciplinary Center for Medical Research of Franceville (CIRMF). These strains were isolated in a previous study from the diarrhoeal stools of 160 children aged 0 to 5 years in the city of Makokou, in accordance with the recommendations of the hospital ethics committee of the decree PROT No. 0020/2015/SG/CNE of the Gabon National Ethics Committee for Research and the Ministry of Health. The isolates were obtained from stool samples collected at the Omar BONGO ONDIMBA Regional Hospital Center in Makokou (CHROBOM), during various collection campaigns from 18 April to 18 October 2023, and from 27 June to 27 December 2024.

The samples were collected from the children after obtaining informed consent from their parents. Phenotypic and molecular tests were performed in January 2025 at the bacteriology laboratory of the Medical Analysis Research Unit, Interdisciplinary Center for Medical Research of Franceville (CIRMF).

2.2. Phenotypic Evaluation of Campylobacter Susceptibility to Fluoroquinolones (FQs)

The evaluation of *Campylobacter* susceptibility to Fluoroquinolones (FQs) was carried out using the antibiotic disc diffusion technique on Mueller–Hinton (MH) agar according to the Kirby–Bauer method. Three molecules from this family were tested: ciprofloxacin (CIP, 5 µg), levofloxacin (LVX, 5 µg), and pefloxacin (PEF, 5 µg) (Liofilchem, Roseto degli Abruzzi, Italy). These represent 2nd and 3rd generation FQs. The standardized inocula at 0.5 McFarland were prepared from pure colonies aged 18 to 24 h in sterile saline (0.9% NaCl), then inoculated onto the surface of Mueller–Hinton-F agar medium supplemented with 5% mechanically defibrinated horse blood and 20 mg/L of β-NAD used for slow-growing bacteria, and all incubated at 35 ± 2 °C in microaerophilic conditions for 18 to 24 h. Finally, the interpretation of the results was carried out in accordance with the standards of the Antibiogram Committee of the French Society of Microbiology (CASFM): recommendations 2024 v.1.0.

2.3. Molecular Research of Fluoroquinolone Resistance Genes

The total DNA extraction was performed from colonies less than 48 h old from a fresh culture obtained after incubation on solid medium. Total DNA from all isolates was extracted according to the instructions of the Invitrogen Purlink DNA/RNA extraction kit (Thermo Fisher Scientific, Waltham, MA, USA). Specific primers (Table 1) were used for the detection of the chromosomal *gyrA* gene coding for gyrase and those of the *qnr* complex (*qnrA*, *qnrB*, and *qnrS*) coding for plasmid-mediated resistance to FQs [10]. The PCR assay for *gyrA* was designed to detect the presence of the gene itself, rather than specific resistance-conferring point mutations within the QRDR.

Table 1. Primers used for the genotypic detection of quinolone resistance.

Genes	Primers	Primer Sequence (5′–3′)	Size (bp)
gyrA	*gyrA-F*	AACCGTTGATGACTTCCGTC	511
	gyrA-R	TATGGCGATGTCGCTCATTTG	
qnrA	*qnrA-F*	ATTTCTCACGCCAGGATTTG	516
	qnrA-R	GATCGGCAAAGGTTAGGTCA	
qnrB	*qnrB-F*	GATCGTGAAAGCCAGAAAGG	383
	qnrB-R	ACGATGCCTGGTAGTTGTCC	
qnrS	*qnrS-F*	ACGACATTCGTCAACTGCAA	417
	qnrS-R	TAAATTGGCACCCTGTAGGC	

The amplification was performed as described in a previous study [11]. The different multiplex and simplex PCR reactions were carried out in a final reaction volume of 20 µL, containing the primer mix concentrated at 0.3µM from the *qnr* complex, the 1X multiplex master mix (Qiagen® Multiplex PCR kit, Hilden, Germany) and 2 µL of diluted DNA matrix for the multiplex PCR. In contrast, for the simplex PCR of the *gyrA* gene, the PCR mix consisted of 10 µL of 2X master mix (Amplitaq Golden, Foster, CA, USA), DgyrA primers concentrated at 0.5µM, and the same volume of diluted DNA. The amplification was carried out using a thermocycler (Bio-RAD, T100TM, Hercules, CA, USA) under the following conditions: the initial denaturation step was performed at 95 °C for 15 min, followed by 30 cycles comprising a denaturation step at 94 °C for 1.5 min, a hybridization step at 54 °C for 1 min, an elongation step at 72 °C for 1 min, and a final elongation step at 72 °C for 7 min.

The PCR products were separated by electrophoresis on a 1.5% (*w*/*v*) agarose gel, prepared in a 0.5X TBE solution containing 0.5 µg mL^{-1} EtBr. The electrophoresis was carried out at a voltage of 100 V and a current of 100 mA for 1.5 h using a CONSORT EV243 (Power Supply, Consort nv, Turnhout, Belgium) device. The gel was visualized using the Quantum ST4 1100/26 MX software (Quantum, Burladingen, Baden-Württemberg, Germany).

3. Results

3.1. Phenotypic Evaluation of Campylobacter Susceptibility to Fluoroquinolones (FQs)

The FQ susceptibility profiles are presented in Figure 1.

These results show that all *Campylobacter* isolates are resistant to fluoroquinolones with a very high prevalence of 100% (116/116). This prevalence of resistance is the same for ciprofloxacin (100%, *n* = 116/116), levofloxacin (100%, *n* = 116/116), and pefloxacin (100%, *n* = 116/116), which are second-, and third-generation fluoroquinolones.

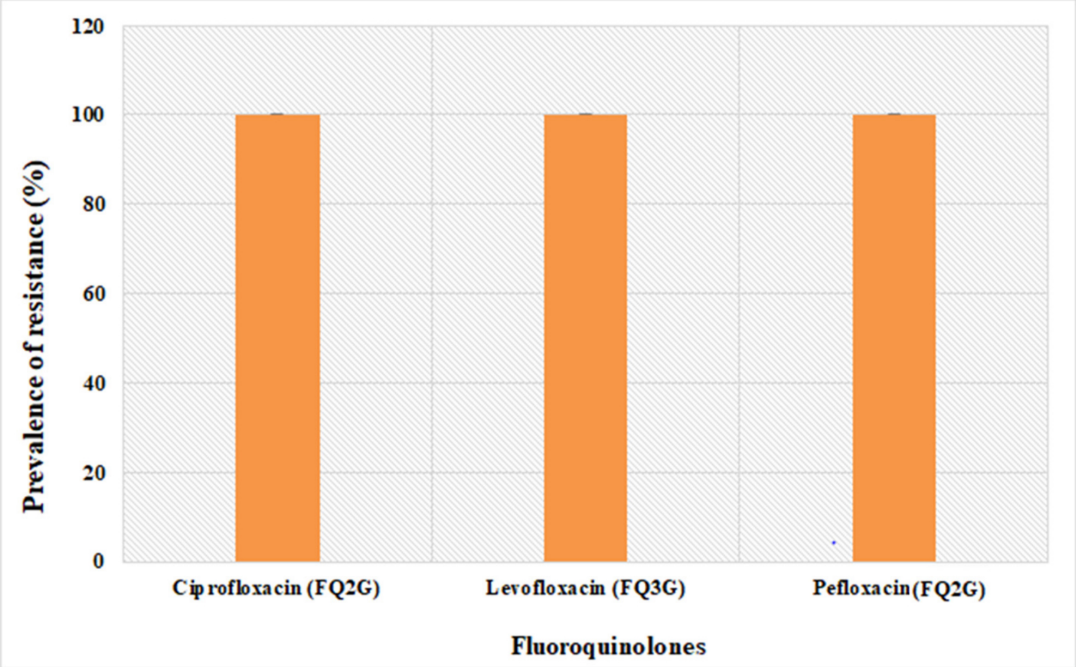


Figure 1. Prevalence of *Campylobacter* resistance to fluoroquinolones. FQ2G: second-generation fluoroquinolones; FQ3G: third-generation fluoroquinolones.

3.2. Molecular Determinism of Fluoroquinolone Resistance in Campylobacter Isolates

The PCR search for the genes *qnrA*, *qnrB*, *qnrS*, and *gyrA* recorded prevalences of 0.0% (0/116), 4.3% (5/116), 51.7% (60/116), and 94.0% (109/116), respectively (Table 2). Three isolates, or 2.6% (3/116), showed no detection of the target genes. The results illustrating the molecular basis for FQ resistance are shown in Table 2.

Table 2. Distribution of fluoroquinolone genes.

	Campylobacter	
	Effective (*n* = 116)	**Percentage (%)**
Mono-detection	52	44.8
gyrA	48	41.4
qnrA	0	0.0
qnrS	4	3.4
qnrB	0	0
Co-detection	61	52.6
gyrA-qnrS	56	48.3
gyrA-qnrB	5	4.3

The results show that, out of the 116 *Campylobacter* isolates, 61 (52.6%) carry at least one of the three tested *qnr* genes in addition to the *gyrA* gene. On the other hand, four isolates carry only the *qnrS* gene (3.4%), and 41.4% (48/116) carry the *gyrA* gene alone (Table 2).

Moreover, the results show co-detection with *gyrA*, with the most predominant being obtained with the *qnrS* gene (48.3%), followed by the *qnrB* gene (4.3%) (Figure 2).

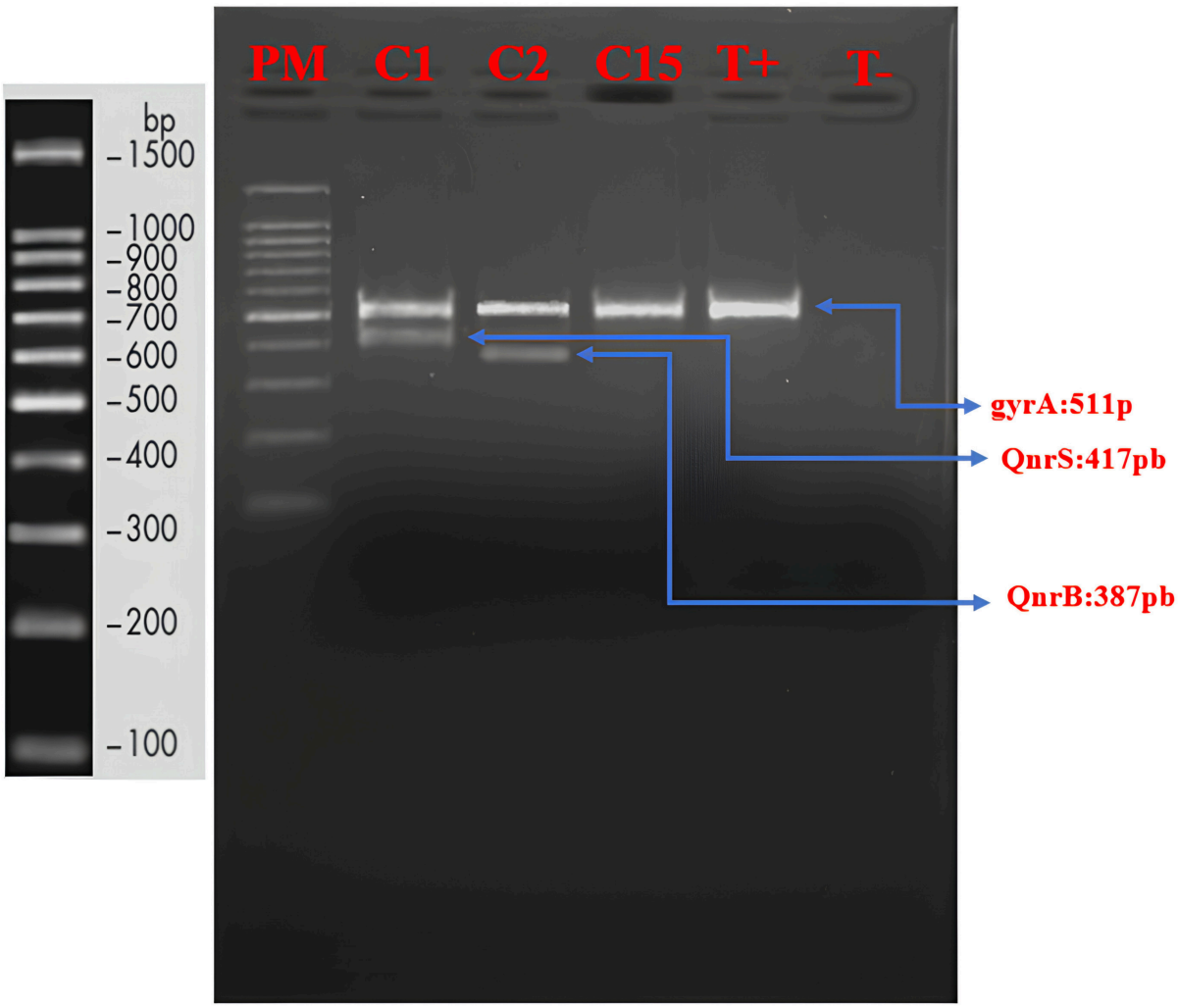


Figure 2. PCR profiles of selected *C. jejuni* isolates on 1.5% agarose gel. PM: molecular weight marker (1500 bp); T^+: positive control; T^-: negative control; C: *C. jejuni*.

The results in Table 3 reveal that the prevalence of plasmid-mediated quinolone resistance (PMQR) is high for the species *C. jejuni* (62.2%), *C. lari* (50%), *C. coli* (45.83%), and moderate for *C. spuctorum* (28.57%).

Table 3. Summary of the phenotypic and genotypic resistance of *Campylobacter* isolates to fluoroquinolones.

		Phenotypic Detection			**Genetic Determinants**			
		Generation of FQ			**Chromosomal**	**Plasmidic**		
		Ciprofloxacine (FQ2G)	**Lévofloxacine (FQ3G)**	**Pefloxacine (FQ2G)**	***gyr A***	***qnrA***	***qnrB***	***qnrS***
Strains	**Effective**	**Resistance *n* = Effective (%)**	**Resistance *n* = Effective (%)**	**Resistance *n* = Effective (%)**	**Present *n* = Effective (%)**	**Present *n* = Effective (%)**	**Present *n* = Effective (%)**	**Present *n* = Effective (%)**
C. coli	24	24 (100%)	24 (100%)	24 (100%)	23 (95.83%)	0 (0%)	1 (4.17%)	10 (41.67%)
C. lari	2	2 (100%)	2 (100%)	2 (100%)	1 (50%)	0 (0%)	0 (0%)	1 (50%)
C. jejunii	83	83 (100%)	83 (100%)	83 (100%)	79 (96.34%)	0 (0%)	3 (3.66%)	48 (58.54%)
C. spuctorum bv feacalis	7	7 (100%)	7 (100%)	7 (100%)	6 (85.71%)	0 (0%)	1 (14.28%)	1 (14.28%)
		100% (116/116)	100% (116/116)	100% (116/116)	94% (109/116)	0% (0/116)	4.3% (5/116)	51.7% (60/116)

4. Discussion

Infectious diarrhoeal caused by *Campylobacter* spp. has been described worldwide as an important gastrointestinal disease. In this study, we investigated the phenotypic susceptibility of *Campylobacter* spp. isolated from diarrhoeal stools of children under 5 years of age in Makokou, Gabon, with a particular focus on fluoroquinolones (FQs), and explored selected genetic determinants potentially associated with FQ resistance. The most striking finding was the complete phenotypic resistance of the isolates to all tested FQs, together with the detection of plasmid-mediated quinolone resistance (PMQR) genes in a substantial proportion of isolates.

The 100% resistance rate observed in this study indicates a very high level of loss of FQ activity among *Campylobacter* isolates circulating in this population. Although such a high prevalence is concerning, it should be interpreted in the context of the relatively small and geographically restricted study population. Nevertheless, the finding is consistent with the high FQ resistance reported in several settings and supports the growing evidence that ciprofloxacin and related FQs have limited utility against *Campylobacter* in many regions [1,3,12]. In contrast, substantially lower ciprofloxacin resistance rates have been reported in Rwanda and other African settings, including 32% reported by Gahamanyi et al. and 33.3% by Worku et al., while Iweriebor et al. reported resistance rates of 20.3% to ciprofloxacin and 19% to norfloxacin in South Africa [2,3,12]. Differences between studies may reflect geographical variation, species distribution, antimicrobial exposure, sampling populations, antimicrobial susceptibility testing methods, and the selective pressures exerted by antimicrobial use in human and animal populations.

The exceptionally high FQ resistance observed in Makokou is epidemiologically relevant because *Campylobacter* is a zoonotic pathogen with important animal reservoirs, particularly poultry, and resistance may be selected in both human and veterinary settings.

Antimicrobial exposure is a recognised driver of FQ resistance in *Campylobacter*, and resistant strains may subsequently disseminate through food chains and environmental pathways [7,13,14].

Similarly, this would undoubtedly be due to acquisition and distribution, but also to the significant role of horizontal transfer of FQ resistance genes [1]. Indeed, numerous studies have shown that the widespread acquisition of resistance to FQs is the result of spontaneous independent mutations and is accelerated by horizontal DNA transfer, conferring resistance between strains of members of the *Campylobacter* genus and other bacteria. Also, from a mechanistic perspective, FQ resistance in *Campylobacter* differs from that observed in many other Gram-negative bacteria. *Campylobacter* lacks the canonical *parC* and *parE* genes encoding topoisomerase IV [14,15], and resistance to FQs is primarily associated with mutations within the quinolone-resistance-determining region (QRDR) of *gyrA*. The C257T nucleotide substitution, resulting in the Thr86Ile (T86I) amino acid substitution, is the most frequently reported alteration associated with FQ resistance in *Campylobacter*. Other substitutions, including T86K, A70T and D90N, have also been described. Importantly, a single QRDR mutation may substantially reduce FQ susceptibility, distinguishing *Campylobacter* from organisms in which resistance commonly involves alterations in both DNA gyrase and topoisomerase IV [15,16].

In addition to target-site modification, the CmeABC multidrug efflux system plays an important complementary role in FQ resistance. CmeABC reduces intracellular antimicrobial accumulation and can act synergistically with *gyrA* mutations to increase the level of resistance and facilitate the emergence of resistant mutants [14,17]. Moreover, this same study highlights that the inhibition of this efflux pump can prevent the emergence of clinically relevant *Campylobacter* mutants resistant to FQs [17]. Therefore, the coexistence of QRDR mutations and enhanced efflux activity represents a biologically plausible expla-

nation for high-level FQ resistance in *Campylobacter*. In contrast, Sharifi's work shows a significant difference in the expression of the *cmeA* gene between ciprofloxacin-resistant and susceptibility *Campylobacter* isolates, highlighting the crucial contribution of the CmeABC efflux pump in FQ resistance [18].

In the present study, *gyrA* was successfully amplified in most phenotypically FQ-resistant isolates. However, amplification of *gyrA* alone cannot establish whether resistance-associated mutations are present, because *gyrA* is an intrinsic chromosomal gene in susceptible as well as resistant strains. Consequently, our molecular results cannot be interpreted as evidence that *gyrA* is not involved in the observed resistance. Sequencing of the QRDR, particularly the region encompassing codon 86, would be required to determine whether the canonical C257T/T86I mutation or other resistance-associated substitutions are present. This represents an important limitation of the current study and should be addressed in future investigations.

A particularly noteworthy finding was the detection of *qnrS* in 48.3% of isolates and *qnrB* in 4.3% of isolates. This study, to our knowledge, is the first report from Gabon describing the detection of these PMQR determinants in *Campylobacter* isolates recovered from diarrhoeal stools in children under 5 years old. These results reinforce the hypothesis of the involvement of plasmid-mediated *qnr* genes in FQ resistance. The prevalence of *qnrS* observed here is comparable to that reported by Gharbi et al. in animal samples, whereas the prevalence of *qnrB* was substantially lower [19]. The occurrence of these determinants in isolates from both human and animal sources raises the possibility of a broader ecological distribution of quinolone-resistance determinants. In addition, it has been shown that the presence of *qnr* genes is associated with a low level of resistance to FQs, while other studies highlight that their presence improves the selection of chromosomal mutations leading to high levels of resistance [20]. This would clearly contribute to explaining the total resistance recorded against FQs in this work.

The detection of *qnrS* and *qnrB* is also of particular interest because PMQR determinants have not traditionally been considered established mechanisms of FQ resistance in *Campylobacter*. Previous reviews have identified QRDR mutations in *gyrA* and the CmeABC efflux system as the principal mechanisms of FQ resistance in this genus, while *qnr*, *aac(6′)-Ib-cr* and *qepA* have historically not been demonstrated as established *Campylobacter* resistance determinants [14,16]. Thus, the relatively high frequency of *qnrS* detected in our collection should be interpreted cautiously. PCR amplification alone does not establish that these genes are functional, located on transferable plasmids, or responsible for the resistant phenotype. Confirmation through more sophisticated analyses would substantially strengthen this finding. Although the *qnrA* gene has been reported in several Gram-negative bacteria, it appears to be uncommon or absent in most published *Campylobacter* collections, whereas *qnrB* and particularly *qnrS* have been detected more frequently in the few available studies investigating PMQR determinants in this genus [11,19,21].

The absence of amplification of the investigated resistance determinants in a small proportion of phenotypically resistant isolates further supports the likelihood of additional mechanisms or genetic variation not captured by the molecular targets investigated. These results clearly support that the resistance of diarrhogenic *Campylobacter* to quinolones is not primarily linked to this determinant. The absence of amplification of the tested genes in 2.56% of *Campylobacter* isolates that are resistant to FQs could be explained by the presence of other genetic determinants or mechanisms that were not investigated in this study. In Gram-negative bacteria, additional determinants include the *aac(6′)-lb-cr* genes, which code for an acetyltransferase that acetylates FQs, and the *qepA* and *oqxAB* genes, which code for efflux pumps and other resistance mechanisms [22,23]. Other mechanisms, including

CrpP-like determinants, which code for a phosphorylase, have also been proposed, although their contribution to clinically relevant fluoroquinolone resistance remains debated [22,24].

5. Conclusions

In conclusion, this work has provided an overview of the susceptibility of *Campylobacter* isolates to fluoroquinolones in the city of Makokou. Overall, the 100% FQ resistance observed among *Campylobacter* isolates from children with diarrhoea in Makokou represents an important local AMR signal. The detection of *qnrS* and *qnrB* is particularly noteworthy because PMQR determinants are not traditionally recognised as established mechanisms of FQ resistance in *Campylobacter*. Rather than attributing the phenotype to a single mechanism, our findings support a multifactorial model in which chromosomal target-site mutations and efflux mechanisms may play the dominant role, with the potential contribution of PMQR determinants requiring further genomic and functional confirmation. These results highlight the need to strengthen laboratory-based AMR surveillance and One Health monitoring in Gabon and to generate molecular data capable of distinguishing locally emerging resistance mechanisms from those already established in the regional *Campylobacter* population.

Author Contributions: Conceptualization, J.F.Y. and R.M.M.; methodology, J.F.Y. and O.Z.M.; validation, J.F.Y., R.M.M. and F.M.; formal analysis, O.Z.M., L.F.O., J.-M.A.P. and J.A.O.-N.; investigation, O.Z.M. and J.-M.A.P.; resources, J.-M.A.P. and J.A.O.-N.; data curation, O.Z.M., E.M.N. and F.M.; writing—original draft preparation, J.F.Y., O.Z.M., and R.M.M.; writing—review and editing, J.F.Y., R.M.M., O.Z.M., and F.M.; visualization, J.F.Y., O.Z.M., E.M.N. and F.M.; supervision, J.F.Y.; project administration, J.F.Y. and J.-M.A.P. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Data is contained within the article. The original contributions presented in this study are included in the article. Further inquiries can be directed to the corresponding author.

Acknowledgments: The authors are also grateful to all patients who provided agreement to participate in the study. During the preparation of this manuscript, the authors used Gemini and ChatGPT-4o to correct translation errors. The authors have reviewed and edited the output and take full responsibility for the content of this publication.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

AMR	Antimicrobial resistance
FQ	Fluoroquinolones
FQ2G	Second-generation fluoroquinolones
FQ3G	Third-generation fluoroquinolones
PMQR	Plasmid-mediated quinolone resistance
QRDR	Quinolone resistance determining region
MDR	Multidrug-resistant
XDR	Extensively drug-resistant.
PDR	Pandrug-resistant.

References

1. Sproston, E.L.; Wimalarathna, H.M.L.; Sheppard, S.K. Trends in fluoroquinolone resistance in *Campylobacter*. *Microb. Genom.* **2018**, *4*, e000198. [CrossRef] [PubMed]
2. Iweriebor, B.C.; Abraham, O.S.; Obi, C.L. Evaluation of the incidence of *Campylobacter* species in stool samples of diarrhoeal patients attending selected hospitals in the Eastern Cape Province, South Africa. *Acta Microbiol. Bulg.* **2025**, *41*, 48–56. [CrossRef]
3. Gahamanyi, N.; Habimana, A.M.; Harerimana, J.P.; Iranzi, F.; Ntwali, S.; Kamaliza, G.; Mukayisenga, J.; Bosco, S.J.; Komba, E.V.; Rujeni, N.; et al. High prevalence of antibiotic resistant *Campylobacter* among patients attending clinical settings in Kigali, Rwanda. *BMC Infect. Dis.* **2025**, *25*, 225. [CrossRef] [PubMed]
4. Riedel, C.; Förstner, K.U.; Püning, C.; Alter, T.; Sharma, C.M.; Gölz, G. Differences in the transcriptomic response of *Campylobacter coli* and *Campylobacter lari* to heat stress. *Front Microbiol.* **2020**, *11*, 523. [CrossRef] [PubMed]
5. Paintsil, E.K.; Masanta, W.O.; Dreyer, A.; Ushanov, L.; Smith, S.I.; Frickmann, H.; Zautner, A.E. *Campylobacter* in Africa—A specific viewpoint. *Eur. J. Microbiol. Immunol.* **2023**, *13*, 107–124. [CrossRef] [PubMed]
6. Ma, H.; Su, Y.; Ma, L.; Ma, L.; Li, P.; Du, X.; Gölz, G.; Wang, S.; Lu, X. Prevalence and Characterization of *Campylobacter jejuni* Isolated from Retail Chicken in Tianjin, China. *J. Food Prot.* **2017**, *80*, 1032–1040. [CrossRef] [PubMed]
7. Zachariah, O.H.; Lizzy, M.A.; Rose, K.; Angela, M.M. Multiple drug resistance of *Campylobacter jejuni* and *Shigella* isolated from diarrhoeic children at Kapsabet County referral hospital, Kenya. *BMC Infect. Dis.* **2021**, *21*, 4–11. [CrossRef] [PubMed]
8. Cooper, K.K.; Mourkas, E.; Schiaffino, F.; Parker, C.T.; Vasquez, T.N.P.; Garcia Bardeles, P.F.; Yori, P.P.; Olortegui, M.P.; Villanueva, K.M.; Cachique, L.R.; et al. Sharing of cmeRABC alleles between *C. coli* and *C. jejuni* associated with extensive drug resistance in *Campylobacter* isolates from infants and poultry in the Peruvian Amazon. *mBio* **2025**, *16*, e0205424. [CrossRef] [PubMed]
9. Magiorakos, A.; Srinivasan, A.; Carey, R.B.; Carmeli, Y.; Falagas, M.E.; Giske, C.G.; Harbarth, S.; Hindler, J.F.; Kahlmeter, G.; Olsson-Liljequist, B.; et al. Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: An international expert proposal for interim standard definitions for acquired resistance. *Clin. Microbiol. Infect.* **2012**, *18*, 268–281. [CrossRef] [PubMed]
10. Ranjbar, R.; Behnood, V.; Memariani, H.; Najafi, A.; Moghbeli, M.; Mammina, C. Molecular characterisation of quinolone-resistant Shigella strains isolated in Tehran, Iran. *J. Glob. Antimicrob. Resist.* **2016**, *5*, 26–30. [CrossRef] [PubMed]
11. Mabika Mabika, R.; Mounioko, F.; Souza, A.; Yala, J.F. Phenotypic and molecular characterization of quinolone resistance of enteropathogens isolated from diarrhea in young children in Koula-Moutou/Gabon. *Egypt. J. Basic Appl. Sci.* **2021**, *8*, 345–353. [CrossRef]
12. Worku, M.; Tessema, B.; Ferede, G.; Ochieng, L.; Leliso, S.A.; Mutua, F.; Moodley, A.; Grace, D.; Gelaw, B. *Campylobacter jejuni* and *Campylobacter coli* infection, determinants and antimicrobial resistance patterns among under-five children with diarrhea in Amhara National Regional State, Northwest Ethiopia. *PLoS ONE* **2024**, *19*, e0304409. [CrossRef] [PubMed]
13. Gahamanyi, N.; Mboera, L.E.G.; Matee, M.I.; Komba, E.V.G. Prevalence, risk factors, and antimicrobial resistance profiles of thermophilic *Campylobacter* species in humans and animals in sub-Saharan Africa: A Systematic Review. *Int. J. Microbiol.* **2020**, *2020*, 2092478. [CrossRef] [PubMed]
14. Iovine, N.M. Resistance mechanisms in *Campylobacter jejuni*. *Virulence* **2013**, *4*, 230–240. [CrossRef] [PubMed]
15. Shen, Z.; Wang, Y.; Zhang, Q.; Shen, J. Antimicrobial resistance in *Campylobacter* spp. *Microbiol. Spectr.* **2018**, *2*, 1–13. [CrossRef] [PubMed]
16. Park, M.; Kim, J.; Feinstein, J.; Lang, K.S. Development of fluoroquinolone resistance through antibiotic tolerance in *Campylobacter jejuni*. *Microbiol. Spectr.* **2022**, *10*, e0166722. [CrossRef] [PubMed]
17. Yan, M.; Sahin, O.; Lin, J.; Zhang, Q. Role of the CmeABC efflux pump in the emergence of fluoroquinolone-resistant *Campylobacter* under selection pressure. *J. Antimicrob. Chemother.* **2006**, *58*, 1154–1159. [CrossRef] [PubMed]
18. Sharifi, S.; Bakhshi, B.; Najar, S. Significant contribution of the CmeABC Efflux pump in high-level resistance to ciprofloxacin and tetracycline in *Campylobacter jejuni* and *Campylobacter coli* clinical isolates. *Ann. Clin. Microbiol. Antimicrob.* **2021**, *20*, 36. [CrossRef] [PubMed]
19. Gharbi, M.; Tiss, R.; Chaouch, M.; Hamrouni, S.; Maaroufi, A. Emergence of Plasmid-Mediated Quinolone Resistance (PMQR) genes in *Campylobacter coli* in Tunisia and Detection of New. *Antibiotics* **2024**, *13*, 527. [CrossRef] [PubMed]
20. Yanat, B.; Rodríguez-Martínez, J.; Touati, A. Plasmid-mediated quinolone resistance in *Enterobacteriaceae*: A systematic review with a focus on Mediterranean countries. *Eur. J. Clin. Microbiol. Infect. Dis.* **2017**, *36*, 421–435. [CrossRef] [PubMed]
21. Jacoby, G.A.; Strahilevitz, J.; Hooper, D.C. Plasmid-mediated quinolone resistance. *Microbiol. Spectr.* **2014**, *2*, 1–42. [CrossRef] [PubMed]
22. Ruiz, J. Transferable mechanisms of quinolone resistance from 1998 Onward. *Clin. Microbiol. Rev.* **2019**, *32*, e00007–e00019. [CrossRef] [PubMed]

23. Al-Khafaji, N.S.K.; Almjalawi, B.S.A.; Ewadh, R.M.J.; Al-Dahmoshi, H.O.M.; Abed, S.Y.; Nasrolahi, A.; Nwobodo, D.C.; Kanaan, M.H.G.; Abdullah, S.S.; Saki, M. Prevalence of plasmid-mediated quinolone resistance genes and biofilm formation in different species of quinolone-resistant clinical *Shigella* isolates: A cross-sectional study. *Eur. J. Med. Res.* **2024**, *29*, 419. [CrossRef] [PubMed]
24. Rodríguez-Martínez, J.M.; Machuca, J.; Cano, M.E.; Calvo, J.; Martínez-Martínez, L.; Pascual, A. Plasmid-mediated quinolone resistance: Two decades on. *Drug Resist. Updates* **2016**, *29*, 13–29. [CrossRef] [PubMed]

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.