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Epidemiology, Antimicrobial Resistance, and Molecular Characterisation of Campylobacter Isolates from Diarrheic Patients and Poultry in a Rural Sub-Saharan African Setting

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Abstract

Background: Campylobacteriosis is a leading cause of bacterial gastroenteritis worldwide, with poultry serving as the primary zoonotic reservoir. In sub-Saharan Africa, the burden is compounded by limited sanitation, widespread backyard poultry keeping, and weak antimicrobial resistance (AMR) surveillance. The prevalence, demographic distribution, and homestead risk factors for *Campylobacter* infection among diarrheic patients at Busia County Hospital, western Kenya, have been documented in a preceding publication from this dataset (Ouko *et al.*) [1]. Building on those findings, this study extends the analysis to characterize antimicrobial resistance (AMR) profiles and molecular epidemiology of *Campylobacter* isolates from diarrheic patients and poultry in Busia County, western Kenya. **Methods:** Antimicrobial susceptibility testing was performed on 114 isolates (98 *C. jejuni*, 16 *C. coli*) from human and poultry sources against 11 antibiotics using disc diffusion and broth microdilution methods. Multilocus sequence typing (MLST) was conducted using published seven-locus *Campylobacter* MLST scheme on 87 representative isolates based on AMR phenotype and resistance marker profiles. Genotypic resistance determinants were identified through whole-genome sequencing (WGS) of 24 selected isolates representing each identified MLST profile. **Results:** Resistance to tetracycline (100%) and trimethoprim-sulfamethoxazole (95% - 100%) was universal across all isolate groups. Erythromycin resistance was 57.1% in human *C. jejuni* but absent in poultry *C. jejuni* (0%). Human *C.*

coli exhibited extensively drug-resistant (XDR) profiles, with resistance exceeding 50% in 10 of 11 antibiotics. MLST identified predominant lineages (ST-353, ST-1036, ST-1932) shared between human and poultry isolates, suggesting a possible zoonotic transmission pathway. Isolates belonging to ST-1038 and ST-7784 were unassigned to known clonal complexes, suggesting the need for further evaluation as potential novel African lineages. **Conclusions:** This study documents substantial *Campylobacter* AMR burden in western Kenya. Universal tetracycline and sulfonamide resistance renders these agents therapeutically obsolete for use in clinical scenarios. The emergence of XDR *C. coli* and evidence of shared ST and clone types between humans and animals suggest a possible zoonotic transmission pathway. These data demand urgent One Health interventions, including an enhanced AMR surveillance, a robust antimicrobial stewardship program, and targeted sanitation improvements to reduce infection and transmission rates.

Keywords

Campylobacteriosis, Antimicrobial Resistance (AMR), Multilocus Sequence Typing (MLST), Zoonotic Transmission, One Health, Sub-Saharan Africa

1. Introduction

In sub-Saharan Africa, the epidemiological burden of campylobacteriosis is amplified by structural determinants, including limited access to safe water, inadequate sanitation infrastructure, and widespread backyard poultry husbandry with close human-animal contact. These factors facilitate the easier spread of enteric diseases in countries with fragile health systems that have minimal capacity for enteric disease surveillance [2]. A pooled prevalence of 9.9% (95% CI: 8.4% - 11.6%) has been reported across 20 sub-Saharan African countries, though this likely underestimates the true burden given diagnostic limitations and under-reporting [3]. In Kenya specifically, *Campylobacter* has been identified as a leading bacterial pathogen among diarrheic children, with prevalence estimates ranging from 7.1% to 20.3% depending on the study population and diagnostic methodology [4]. Despite these vulnerabilities, microbiological data documenting species-specific prevalence, zoonotic transmission pathways, phylogenetic diversity of isolates, and antimicrobial resistance (AMR) patterns in the region remain sparse and geographically fragmented [5]. A preceding study from this same dataset characterized the prevalence and homestead risk factors for *Campylobacter* infection among 1200 diarrheic patients at Busia County Hospital, western Kenya, documenting an overall isolation rate of 11.6%, the predominance of *C. jejuni* (89.2%), and poultry farming as the leading risk factor across all age groups [1].

The emergence and global dissemination of antimicrobial-resistant *Campylobacter* strains represent an escalating threat to clinical management and public

health. Fluoroquinolones and macrolides, the antimicrobials of choice for severe campylobacteriosis, are increasingly compromised by rising resistance rates driven by veterinary antibiotic use, particularly in poultry production, and subsequent zoonotic transmission to humans [6]. Resistance to fluoroquinolones is primarily mediated by point mutations in the quinolone resistance-determining region (QRDR) of *gyrA*, while macrolide resistance involves 23S rRNA mutations or the horizontally acquired *erm*(B) methylase gene [7] [8]. Tetracycline resistance, mediated by the plasmid-borne ribosomal protection protein *tet*(O), has reached near-universal prevalence in many low- and middle-income settings [9]. The convergence of these resistance mechanisms within individual strains has given rise to multidrug-resistant (MDR) and extensively drug-resistant (XDR) *Campylobacter* phenotypes that severely constrain therapeutic options [10]. Recognising this trajectory, the World Health Organization classified fluoroquinolone-resistant *Campylobacter* among the high-priority pathogens requiring urgent research and development of new antimicrobials [11].

Molecular epidemiological tools have substantially advanced our understanding of *Campylobacter* population structure, host association, and resistance gene dissemination. Multilocus sequence typing (MLST), based on allelic variation at seven housekeeping loci, enables standardized genotyping and source attribution by linking human clinical isolates to specific animal reservoirs through shared clonal complexes [12] [13]. MLST-based source attribution studies have consistently implicated poultry-associated clonal complexes, particularly CC-21, CC-45, CC-257, CC-353, and CC-828, as the predominant contributors to human campylobacteriosis [14]-[16]. More recently, whole-genome sequencing (WGS) has provided enhanced resolution for tracking resistance gene flow, identifying novel genomic islands, and resolving transmission networks at the strain level [13]. However, molecular data from sub-Saharan African settings remain disproportionately underrepresented in global databases such as PubMLST, limiting the resolution of regional transmission networks and hampering the development of context-specific control strategies [3].

Building on the epidemiological and risk factor analyses reported by Ouko *et al*. [1], this study aimed to: 1) characterise the antimicrobial resistance profiles and minimum inhibitory concentration (MIC) distributions of *Campylobacter* isolates from clinical and poultry sources in western Kenya; and 2) investigate the molecular epidemiology, clonal structure, and genotypic resistance determinants of circulating *Campylobacter* sequence types using MLST. Together, these objectives provide a comprehensive One Health perspective on campylobacteriosis AMR dynamics at the human-poultry interface in a sub-Saharan African context, addressing a critical evidence gap for the region.

2. Materials and Methods

2.1. Study Design, Setting, and Population

A cross-sectional study was conducted among 1200 participants seeking out-pa-

tient treatment for acute diarrhea (≥3 loose stools within 24 hours) from February 2017 to April 2019 at participating health facilities in Busia County, western Kenya. This region is characterized by smallholder mixed farming, ubiquitous backyard poultry keeping, and limited veterinary antimicrobial stewardship [1]. Busia County borders Uganda and represents a high-traffic cross-border zone with potential for trans-boundary pathogen dissemination.

Only patients who provided written informed consent were enrolled in the study, while consent was sought from parents or guardians on behalf of participating minors. In addition, participants must not have used antibiotics in the 5 days preceding enrollment. Non-diarrheal cases and those who declined to participate were excluded from the study.

Alongside clinical sampling, poultry isolates were obtained from commercial and backyard poultry sources within the same catchment area to enable comparative molecular analysis across the human-animal interface. Ethical approval was obtained from the Kenya Medical Research Institute review board (IRB number 27/09/2016_3320). The study was conducted in accordance with the Declaration of Helsinki and national research ethics guidelines.

2.2. Bacteriological Isolation and Species Identification

In the original study [1], stool specimens were collected in sterile containers and transported to the laboratory within 4 hours under cold-chain conditions. Specimens were processed using selective enrichment in Bolton broth supplemented with antimicrobial supplements (cefoperazone, vancomycin, trimethoprim, and amphotericin B) at 42˚C under microaerophilic conditions (5% O_2, 10% CO_2, 85% N_2) for 24 hours, followed by subculture onto modified charcoal cefoperazone deoxycholate agar (mCCDA; Oxoid, UK) and incubation at 42˚C under microaerophilic conditions for 48 hours. Presumptive *Campylobacter* colonies; grey, flat, and spreading with a metallic sheen, were confirmed by Gram staining (curved Gram-negative rods), oxidase positivity, catalase production, and absence of aerobic growth at 25˚C. Species-level identification as *C. jejuni* or *C. coli* was performed by multiplex PCR targeting the *hipO* gene (*C. jejuni*-specific hippuricase) and *ask* gene (*C. coli*-specific aspartokinase) [17] [18]. Pure cultures were preserved at −80˚C in Microbank™ beads (Prolab Diagnostics) mixed with sterile cryopreservation media.

2.3. Recovery of Archived Isolates

For the current study, archived isolates were subcultured onto mCCDA (Oxoid, UK) and incubated at 42˚C under microaerophilic conditions for 48 hours. For isolates that failed to grow, recovery was attempted by transferring the archival beads into the selective enrichment Bolton broth enriched with antimicrobials and *campylobacter* growth factors, followed by incubation at 42˚C under microaerophilic conditions for 24 hours before subculturing onto mCCDA (Oxoid, UK).

Of the 124 *C. jejuni* and 15 *C. coli* isolates obtained in the previous study, only

84 *C. jejuni* and 12 *C. coli* were successfully recovered for the current study, with the remainder lost to culture. Similarly, of the original 16 *C. jejuni* and 6 *C. coli* isolates recovered from poultry, only 14 and 4 isolates, respectively, were recovered for the current study. The identity of the recovered isolates was confirmed using PCR schemes as described for the original study.

2.4. Antimicrobial Susceptibility Testing

Antimicrobial susceptibility was determined on the 114 confirmed isolates (98 *C. jejuni*, 16 *C. coli*) using the Kirby-Bauer disc diffusion method on Mueller-Hinton agar supplemented with 5% defibrinated horse blood, incubated at 42˚C under microaerophilic conditions for 24 hours. The following antimicrobial agents representing six pharmacological classes were tested: tetracycline (TET, 30 µg); sulfonamides/diaminopyrimidines, represented by trimethoprim-sulfamethoxazole (SXT, 1.25/23.75 µg); macrolides, represented by erythromycin (ERY, 15 µg); fluoroquinolones, represented by ciprofloxacin (CIP, 5 µg); penicillins and *β*-lactam/*β*-lactamase inhibitor combinations, including ampicillin (AMP, 10 µg) and amoxicillin-clavulanate (AMC, 20/10 µg); and cephalosporins, including cefoxitin (FOX, 30 µg), cefotaxime (CTX, 30 µg), ceftazidime (CAZ, 30 µg), cefuroxime (CXM, 30 µg), and ceftriaxone (CRO, 30 µg). In this panel, ERY, CIP, TET, AMP and AMC were chosen based on their frequent use to treat clinical and veterinary cases, while the rest of the antimicrobial panel was included in order to gain a clearer AMR phenotypic landscape that guided further molecular work on the isolates.

Minimum inhibitory concentrations (MICs) were determined based on broth microdilution method using cation-adjusted Mueller-Hinton broth supplemented with 2.5% lysed horse blood. Custom-designed panels (Sensititre™ EUCAMP2, Thermo Fisher Scientific) or equivalent were used following the EUCAST guidelines for *Campylobacter* (EUCAST, 2023). Results were interpreted according to EUCAST epidemiological cut-off values (ECOFFs) or based on CLSI breakpoints for agents without EUCAST criteria [19]. *Campylobacter jejuni* ATCC 33560 served as the quality control strain. Multidrug resistance (MDR) was defined as acquired non-susceptibility to at least one agent in three or more antimicrobial categories, and extensive drug resistance (XDR) as non-susceptibility to at least one agent in all but two or fewer categories, following internationally standardized definitions [20] [21].

2.5. Detection and Characterization of Resistance Genes

Genomic DNA extracted from each isolate served as template for gene-specific PCR assays targeting chromosomal resistance loci (*gyr*A, the domain V region of 23S rRNA, and the ribosomal protein genes *rplD* and *rplV*) as well as acquired resistance determinants [*tet*(O), *β*-lactam*ases*, and *erm*(B)]. All assays were adapted from previously validated protocols for *Campylobacter* antimicrobial resistance genotyping, as detailed below.

2.5.1. Fluoroquinolone Resistance—*gyrA*

The quinolone resistance-determining region (QRDR) of *gyrA* was interrogated using the mismatch amplification mutation assay (MAMA)-PCR [22], which permits selective amplification of the C257T point mutation encoding the Thr-86-Ile substitution in GyrA; the principal determinant of high-level ciprofloxacin resistance in *C. jejuni* [22] [23]. All amplicons were confirmed by bidirectional Sanger sequencing to discriminate this mutation from other less common QRDR substitutions (e.g., Asp-90-Asn, Ala-70-Thr) [24].

2.5.2. Macrolide Resistance—23S rRNA, *rplD* (L4), and *rplV* (L22)

Domain V of the 23S rRNA gene was amplified using the primer pair 5'-GTAAACGGCG GCCGTAACTA-3' (forward) and 5'-GACCGAACTGTCTCACGACG-3' (reverse) under the conditions described before [25], enabling detection of the A2074C and A2075G transitions (corresponding to *E. coli* positions A2058 and A2059) that confer high-level erythromycin resistance through disruption of macrolide binding in the peptidyl transferase loop [26] [27].

Ribosomal protein genes *rplD* (encoding L4) and *rplV* (encoding L22), in which insertions or amino acid substitutions can contribute to macrolide resistance synergistically with 23S rRNA mutations [25], were amplified with the following primer pairs:

1) *rplD*: 5'-GTAGTTAAAGGTGCAGTACCA-3'/5'-GCGAAGTTTGAATAACTACG-3'

2) *rplV*: 5'-GAATTTGCTCCAACACGC-3'/5'-ACCATCTTGATTCCCAGTTTC-3'

Thermal cycling consisted of initial denaturation at 94˚C for 5 min, followed by 35 cycles of 94˚C for 30 s, 52˚C for 30 s, and 72˚C for 45 s, with a final extension at 72˚C for 10 min [25].

2.5.3. Tetracycline Resistance—*tet*(O)

The ribosomal protection gene *tet*(O) was detected by gene-specific PCR using primer sequences and conditions previously validated in *Campylobacter* resistance surveys [9] [28]-[30]. This gene, which encodes a GTPase that displaces tetracycline from the ribosomal A-site, is the sole determinant of acquired tetracycline resistance documented in *Campylobacter* spp. and may be plasmid- or chromosomally encoded.

2.5.4. β-Lactam Resistance—*bla*$_{\text{OXA-}}$ Genes

The class D β-lactamase gene *blaXA*$_{\text{OXA-}}$ located at the Cj0299 locus, was amplified using primers originally designed by Alfredson and Korolik [31], who first characterized this enzyme from *C. jejuni*, who demonstrated that strain GC015. Primer specificity and the expected 372-bp diagnostic amplicon were validated following the protocol of Griggs *et al*. [32].

2.5.5. Macrolide Resistance by rRNA Methylation—*erm*(B)

The horizontally transferable rRNA methylase gene *erm*(B) was detected using

primers targeting the conserved coding region of the methyltransferase, as originally described by Wang *et al*. [24], who provided the first report of *erm*(B)-mediated macrolide resistance in Campylobacter coli strain ZC113 harbored on a multidrug resistance genomic island. Amplification and sequence confirmation followed the conditions of subsequent surveillance studies that documented *erm*(B) dissemination in both *C. coli* and *C. jejuni* [33] [34].

2.5.6. Amplicon Analysis and Sequence Confirmation

All amplicons were resolved by electrophoresis on 1.5% (w/v) agarose gels stained with ethidium bromide, excised, purified using a commercial gel extraction kit, and subjected to bidirectional Sanger sequencing. Resulting sequences were aligned to the *C. jejuni* NCTC 11168 reference genome (GenBank accession AL111168) using ClustalW to confirm gene identity and identify specific resistance-associated point mutations or insertions.

2.6. Multilocus Sequence Typing and Genotypic Resistance Characterisation

MLST was performed on 87 representative isolates (76 *C. jejuni*, 11 *C. coli*). These isolates were selected based on their phenotypic resistance profiles and source origins. Genomic DNA was extracted using the DNeasy Blood and Tissue Kit (Qiagen, Germany). The seven housekeeping loci of the *Campylobacter* MLST scheme (*aspA*, *glnA*, *gltA*, *glyA*, *pgm*, *tkt*, *uncA*) were amplified by PCR and sequenced bidirectionally using Sanger sequencing [12] [35]. Allelic profiles and sequence types (STs) were assigned by querying the PubMLST *Campylobacter* database (https://pubmlst.org/campylobacter/) [36]. Clonal complex (CC) assignments were determined using the eBURST algorithm and PubMLST definitions.

2.7. Whole-Genome Sequencing of Representative MLST Types

Genotypic resistance determinants were identified through whole-genome sequencing (WGS) of 24 selected isolates representing each identified MLST profile. Libraries were prepared using the Nextera XT DNA Library Preparation Kit (Illumina) and sequenced on the Illumina MiSeq platform, generating 2 × 250 bp paired-end reads. Raw reads were quality-filtered ($Q \geq 30$), assembled *de novo* using SPAdes v3.15, and resistance genes identified by *in silico* screening against the Comprehensive Antibiotic Resistance Database [37] and ResFinder 4.0 [38]. Point mutations in *gyrA* (fluoroquinolone resistance), 23S rRNA (macrolide resistance), and ribosomal protein genes (*rplD*, *rplV*) were identified by alignment against reference sequences (*C. jejuni* NCTC 11168). The presence of *tet*(O), $bla_{\text{OXA-61}}$, and *erm*(B) was confirmed by gene-specific PCR and sequence verification.

2.8. Statistical Analysis

Statistical comparisons of AMR proportions between species (*C. jejuni* vs. *C. coli*) and sources (human vs. poultry) employed Fisher's Exact Test for pairwise com-

parisons and the Mann-Whitney U Test for overall resistance score distributions. Odds ratios (OR) with 95% confidence intervals (CI) were calculated to quantify associations between source/species and resistance phenotypes. Simpson's diversity index (1-D) was used to assess MLST diversity within and between populations. Phylogenetic relationships among STs were visualised using minimum spanning trees generated in PHYLOViZ 2.0 [39]. A two-tailed p-value < 0.05 was considered statistically significant. All analyses were performed using SPSS v26.0 (IBM Corp., Armonk, NY) and R v4.2 as previously described by Borchani *et al.* [40].

3. Results

3.1. Isolation Rates, Demographic Distribution, and Risk Factors: Summary of Previously Published Findings

The isolation rates, species distribution, demographic characteristics, and age-stratified risk and protective factors for *Campylobacter* infection in this patient cohort have been described in detail in Ouko *et al.* [1]. In brief, 139 *Campylobacter* isolates were recovered from 1200 diarrheic patients (overall isolation rate 11.6%), with *C. jejuni* accounting for 89.2% of isolates and *C. coli* for 10.8%. Isolation rates declined progressively with age: children under two years had the highest rate (13.7%), followed by 2 - 5 years (10.2%) and those older than five years (9.4%), though differences across age groups were not statistically significant ($p > 0.05$). Rates were comparable between sexes and between urban and rural residents.

Homestead poultry farming was the most consistent risk factor across all age strata (OR range: 6.47 - 10.05, all $p < 0.001$). Additional age-specific risks included untreated pond water consumption (under-five age groups), contact with domestic pets and chicken meat consumption (2 - 5 years), and raw milk consumption and household contact with a diarrheal person (over-five years). Breastfeeding (OR: 0.24, $p < 0.001$) and regular toilet use (OR: 0.08, $p < 0.001$) were independently protective in children under two years. Urban residence was protective in children aged 2 - 5 years (OR: 0.47, $p = 0.041$). Full details of these findings, including the study design, recruitment strategy, and multivariable logistic regression models, are reported by Ouko *et al.* [1].

3.2. Antimicrobial Resistance Profiles

Antimicrobial susceptibility testing was performed on 114 *Campylobacter* isolates comprising 98 *C. jejuni* (84 human, 14 poultry) and 16 *C. coli* (12 human, 4 poultry). Resistance prevalence varied substantially by species, source, and antimicrobial class (**Table 1**).

3.2.1. Universal Resistance to Tetracycline and Trimethoprim-Sulfamethoxazole

Resistance to tetracycline was observed in 100% of all isolate groups regardless of species or source, representing the most uniformly distributed resistance phenotype in this study (**Table 2**). Trimethoprim-sulfamethoxazole resistance was sim-

ilarly near-ubiquitous, recorded in 95.2% of human *C. jejuni* and 100% of all other groups.

Table 1. Antimicrobial resistance in *C. Jejuni* and *C. coli* obtained from the study population.

Antimicrobial Agent	Class	Human *C. jejuni* (n = 84) n (%)	*Poultry C. jejuni* (n = 14) n (%)	p-value, *C. jejuni*	Human *C. coli* (n = 12) n (%)	Poultry *C. coli* (n = 4) n (%)	p-value, *C. coli* (n = 16)
Tetracycline (TET)	TET	84 (100.0)	14 (100.0)	—	12 (100.0)	4 (100.0)	—
Trimethoprim-sulfamethoxazole (SXT)	FOL	80 (95.2)	14 (100.0)	1	12 (100.0)	4 (100.0)	—
Erythromycin (ERY)	MAC	48 (57.1)	0 (0.0)	<0.001	12 (100.0)	3 (75.0)	0.25
Ciprofloxacin (CIP)	FQ	31 (36.9)	4 (28.6)	0.769	10 (83.3)	2 (50.0)	0.245
Ampicillin (AMP)	PEN	31 (36.9)	14 (100.0)	<0.001	12 (100.0)	4 (100.0)	—
Amoxicillin-clavulanate (AMC)	BL/BLI	7 (8.3)	14 (100.0)	<0.001	9 (75.0)	2 (50.0)	0.547
Cefoxitin (FOX)	CEPH-2	35 (41.7)	14 (100.0)	<0.001	2 (16.7)	2 (50.0)	0.525
Cefotaxime (CTX)	CEPH-3	9 (10.7)	0 (0.0)	0.352	7 (58.3)	3 (75.0)	1
Ceftazidime (CAZ)	CEPH-3	31 (36.9)	0 (0.0)	0.006	10 (83.3)	3 (75.0)	1
Cefuroxime (CXM)	CEPH-2	5 (6.0)	4 (28.6)	0.02	7 (58.3)	4 (100.0)	0.245
Ceftriaxone (CRO)	CEPH-3	18 (21.4)	3 (21.4)	1	10 (83.3)	4 (100.0)	1

Frequency and percentage of antimicrobial resistance among *Campylobacter jejuni* (n = 98) and *Campylobacter coli* (n = 16) isolates stratified by species and source of isolation (human clinical vs. poultry). Antimicrobial agents tested were tetracycline (TET), trimethoprim-sulfamethoxazole (SXT), erythromycin (ERY), ciprofloxacin (CIP), ampicillin (AMP), amoxicillin-clavulanate (AMC), cefoxitin (FOX), cefotaxime (CTX), ceftazidime (CAZ), cefuroxime (CXM), and ceftriaxone (CRO). Agents were grouped into the following antimicrobial classes: tetracyclines (TET), folate pathway inhibitors (FOL), macrolides (MAC), fluoroquinolones (FQ), penicillins (PEN), β-lactam/β-lactamase inhibitor combinations (BL/BLI), second-generation cephalosporins (CEPH-2), and third-generation cephalosporins (CEPH-3). Values are expressed as number resistant (%) out of isolates tested per group. p-values were calculated using Fisher's exact test to compare resistance rates between human and poultry isolates within each species; a dash (—) indicates that statistical comparison was not applicable due to uniform resistance (100%) across both sources. Significance was set at $p < 0.05$.

Table 2. Antimicrobial resistance rates among *C. Jejuni* and *C. coli*.

Antibiotic Class	Combination of Resistance to Different Agents	Human *C. jejun* (%) n = 84	*Poultry C. jejuni* (%) n = 14	Human *C. coli* (%) n = 12	*Poultry C. coli* (%) *n* = 4	Resistance Distribution
Tetracyclines	TET	**100**	**100**	**100**	**100**	All groups
Folate inhibitors	SXT and TRIM	95.2	**100**	**100**	**100**	All groups (except Human CJ)
Macrolides	ERY	57.1	0.0	**100**	**75**	Human *C. coli*
Fluoroquinolones	CIP	36.9	28.6	**83.3**	50	Human *C. coli*
BL/BLI	AMP and AMC	7.0	**100**	**75**	50.0	Poultry *C. jejuni*

Continued

2nd-generation cephalosporins	FOX and CXM	65.0	29.0	58.0	50.0	Poultry *C. coli*
3rd-generation cephalosporins	CTX, CAZ, and CRO	9.0	0.0	7.0	3.0	Poultry C. coli

Resistance to different combinations of antimicrobials (%) among *Campylobacter jejuni* (n = 98) and *Campylobacter coli* (n = 16) isolates from human clinical and poultry sources, stratified by species and host origin. Antimicrobial agents tested included tetracycline (TET), trimethoprim-sulfamethoxazole (SXT), erythromycin (ERY), ciprofloxacin (CIP), ampicillin (AMP), amoxicillin-clavulanic acid (AMC), cefoxitin (FOX), cefuroxime (CXM), cefotaxime (CTX), ceftazidime (CAZ), and ceftriaxone (CRO). Penicillins/BLI denotes penicillin-class agents tested in combination with a β-lactamase inhibitor. β-lactam/β-lactamase inhibitor combinations (BL/BLI).

3.2.2. Macrolide and Fluoroquinolone Resistance

Species-specific differences were most pronounced for erythromycin. Resistance was detected in 57.1% of human *C. jejuni* isolates but was entirely absent in poultry *C. jejuni* (0.0%). In contrast, *C. coli* isolates demonstrated substantially higher macrolide resistance: 100% of human and 75.0% of poultry isolates were erythromycin-resistant. Ciprofloxacin resistance was also markedly higher in *C. coli* than *C. jejuni* in human isolates (83.3% vs 36.9%, p = 0.004), consistent with the overall pattern of elevated resistance in *C. coli*.

3.2.3. β-Lactam and Cephalosporin Resistance

Ampicillin and amoxicillin-clavulanate resistance patterns revealed a striking source-specific contrast in *C. jejuni*: poultry isolates showed 100% resistance to a combination of both agents, compared to only 7% in human *C. jejuni* isolates respectively. All *C. coli* isolates, both human and poultry, were fully resistant to ampicillin (100%). Cephalosporin resistance patterns were more variable. Resistance to third-generation cephalosporins (cefotaxime, ceftazidime, ceftriaxone) was substantially higher in *C. coli* than in *C. jejuni* across all source groups, with ceftriaxone resistance reaching 83.3% in human and 100% in poultry *C. coli* isolates.

3.3. Statistical Comparisons of AMR Profiles

Fisher's Exact Test and Mann-Whitney U Test were used to compare resistance proportions between species and sources. *C. coli* isolates demonstrated significantly higher overall resistance than *C. jejuni* across all 11 antibiotics (mean 78.6% vs 46.5%; median 80.0% vs 36.9%; U = 130.0, p = 0.008). In human isolates, *C. coli* showed significantly higher resistance in 8 of 11 agents, with the most pronounced differences for ampicillin (100.0% vs 36.9%, p < 0.001), amoxicillin-clavulanate (75.0% vs 8.3%, p < 0.001), ceftriaxone (80.0% vs 21.4%, p < 0.001), cefuroxime (60.0% vs 6.0%, p < 0.001), and cefotaxime (60.0% vs 10.7%, p < 0.001).

In poultry isolates, significant differences between species were detected for erythromycin, cefotaxime, ceftazidime, and ceftriaxone (all in favour of higher resistance in *C. coli*), whereas *C. jejuni* poultry isolates showed significantly higher resistance to amoxicillin-clavulanate and cefoxitin (both p = 0.039). Comparison of *C. jejuni* isolates by source (human vs. poultry) revealed that poultry isolates

had significantly higher resistance to ampicillin, amoxicillin-clavulanate, and cefoxitin (all $p < 0.001$), while human isolates showed paradoxically higher erythromycin resistance (57.1% vs 0.0%, $p < 0.001$). No overall significant difference in resistance was detected between human and poultry sources when both species were combined ($U = 209.5$, $p = 0.443$).

Multidrug Resistance Classification

All isolate groups fulfilled the criteria for MDR (resistance to three or more antibiotic classes) (Table 3). Human *C. coli* isolates exhibited the most alarming profile, with resistance exceeding 50% in 10 of 11 individual antibiotics and spanning all six antibiotic classes, consistent with classification as extensively drug-resistant (XDR). Poultry *C. jejuni* demonstrated MDR across four to five classes, primarily attributable to near-universal penicillin, cephalosporin, tetracycline, and sulfonamide resistance. Human *C. jejuni* met the MDR threshold principally through macrolide, tetracycline, and sulfonamide resistance.

Table 3. Distribution of MDR phenotypes in *C. Jejuni* and *C. coli* from humans and poultry.

Isolate Group	n	Antibiotics with >50% Resistance	No. Classes with >50% Mean Resistance	Classification[a]	Classes Implicated
Human *C. jejuni*	84	3/11	3	MDR	MAC, TET, FOL
Poultry *C. jejuni*	14	5/11	4 - 5	MDR	PEN, BL/BLI, CEPH-2, TET, FOL
Human *C. coli*	12	10/11	6	XDR	FQ, MAC, PEN, CEPH, TET, FOL
Poultry *C. coli*	4	8/11	5	MDR	MAC, PEN, CEPH, TET, FOL
Overall	114	—	—	MDR (all groups)	—

Summary of multidrug resistance classification among *Campylobacter jejuni* (n = 98) and *Campylobacter coli* (n = 16) isolates stratified by species and source of isolation. "Antibiotics with >50% Resistance" indicates the number of individual antimicrobial agents (out of 11 tested) for which group-level resistance exceeded 50%. "No. Classes with >50% Mean Resistance" denotes the number of distinct antimicrobial classes in which the mean resistance rate surpassed 50%. Classification was assigned according to the criteria of Magiorakos *et al*. [20]: [a]Multidrug-resistant (MDR), defined as acquired non-susceptibility to at least one agent in three or more antimicrobial categories; and extensively drug-resistant (XDR), defined as non-susceptibility to at least one agent in all but two or fewer antimicrobial categories. Antimicrobial classes are abbreviated as follows: tetracyclines (TET), folate pathway inhibitors (FOL), macrolides (MAC), fluoroquinolones (FQ), penicillins (PEN), β-lactam/β-lactamase inhibitor combinations (BL/BLI), and cephalosporins (CEPH; encompassing second- and third-generation agents). A dash (—) indicates that a summary statistic was not applicable for the pooled "Overall" row.

3.4. Minimum Inhibitory Concentration Distributions

MIC distributions were determined for six antimicrobial agents and provided quantitative characterisation of resistance levels beyond categorical classifications (Table 4).

3.4.1. Tetracycline and Trimethoprim-Sulfamethoxazole

Both agents demonstrated complete population shifts above the susceptibility breakpoints across virtually all groups. Tetracycline MICs ranged from 4 mg/L to 128 mg/L in human *C. jejuni*, with 53.6% of isolates exhibiting MICs of 32 - 128

Table 4. MIC profiles of *C. jejuni* and *C. coli* from human and poultry.

Antimicrobial Agent	Breakpoint (S/R, mg/L)	Source	n	MIC Distribution, mg/L: n (%)	Resistant, n (%)
			Campylobacter jejuni		
Erythromycin	S ≤ 4/R > 4	Human	84	0.25 - 2: 36 (42.9%); 8: 28 (33.3%); 16 - 64: 20 (23.8%)	**48 (57.1%)**
		Poultry	14	0.5 - 2: 14 (100%)	**0 (0%)**
Ciprofloxacin	S ≤ 0.5/R > 0.5	Human	84	0.25: 53 (63.1%); 1: 9 (10.7%); 8 - 128: 22 (26.2%)	**31 (36.9%)**
		Poultry	14	0.25: 10 (71.4%); 16 - 128: 4 (28.6%)	**4 (28.6%)**
Tetracycline	S ≤ 2/R > 2	Human	84	4: 31 (36.9%); 16: 8 (9.5%); 32 - 128: 45 (53.6%)	**84 (100%)**
		Poultry	14	16 - 128: 14 (100%)	**14 (100%)**
Ampicillin	S ≤ 8/R > 8	Human	84	≤4: 53 (63.1%); 16: 16 (19.0%); 32 - 128: 15 (17.9%)	**31 (36.9%)**
		Poultry	14	16 - 64: 14 (100%)	**14 (100%)**
Amoxicillin-clavulanate	S ≤ 8/R > 8	Human	84	≤4: 77 (91.7%); 16 - 32: 7 (8.3%)	**7 (8.3%)**
		Poultry	14	16 - 64: 14 (100%)	**14 (100%)**
Trimethoprim-sulfamethoxazole	S ≤ 2/R > 2	Human	84	≤2: 4 (4.8%); 4 - 8: 80 (95.2%)	**80 (95.2%)**
		Poultry	14	4 - 8: 14 (100%)	**14 (100%)**
			Campylobacter coli		
Erythromycin	S ≤ 8/R > 8	Human	12	16: 3 (25.0%); 32 - 128: 9 (75.0%)	**12 (100%)**
		Poultry	4	≤8: 1 (25.0%); 16 - 64: 3 (75.0%)	**3 (75.0%)**
Ciprofloxacin	S ≤ 0.5/R > 0.5	Human	12	0.25: 2 (16.7%); 8 - 128: 10 (83.3%)	**10 (83.3%)**
		Poultry	4	0.25: 2 (50.0%); 32 - 128: 2 (50.0%)	**2 (50.0%)**
Tetracycline	S ≤ 2/R > 2	Human	12	16 - 128: 12 (100%)	**12 (100%)**
		Poultry	4	16 - 128: 4 (100%)	**4 (100%)**
Ampicillin	S ≤ 8/R > 8	Human	12	16 - 128: 12 (100%)	**12 (100%)**
		Poultry	4	16 - 128: 4 (100%)	**4 (100%)**
Amoxicillin-clavulanate	S ≤ 8/R > 8	Human	12	≤8: 3 (25.0%); 16 - 32: 9 (75.0%)	**9 (75.0%)**
		Poultry	4	≤8: 2 (50.0%); 16 - 32: 2 (50.0%)	**2 (50.0%)**
Trimethoprim-sulfamethoxazole	S ≤ 2/R > 2	Human	12	4 - 8: 12 (100%)	**12 (100%)**
		Poultry	4	4 - 8: 4 (100%)	**4 (100%)**

Breakpoints are expressed in mg/L. **S**, susceptible; **R**, resistant. n = number of isolates tested per source. MIC distribution values are grouped by tested dilution range as reported; percentages are of the source-specific total (n) and may not sum to 100% due to rounding. "Resistant, n (%)" is the sum of isolates with MIC values above the resistance break-point.

mg/L, indicative of high-level resistance mediated by acquired determinants. Similarly, trimethoprim-sulfamethoxazole MICs were uniformly concentrated at 4 - 8 mg/L across all groups, with no susceptible isolates detected in most categories.

3.4.2. Erythromycin

Erythromycin MIC distributions revealed a bimodal pattern in human *C. jejuni*: 42.9% of isolates were susceptible (MIC: 0.25 - 2 mg/L), while 33.3% showed MICs

at 8 mg/L and 23.8% exhibited high-level resistance (MIC: 16 - 64 mg/L). Poultry *C. jejuni* isolates were entirely susceptible (MIC: 0.5 - 2 mg/L), starkly contrasting with the human isolate pattern. Human *C. coli* isolates showed uniformly high MICs, with 75.0% above 32 mg/L, confirming clinically significant macrolide resistance.

3.4.3. Ciprofloxacin

Ciprofloxacin MIC distributions were bimodal across most groups, with resistant populations showing markedly elevated MICs (predominantly 8 - 128 mg/L), suggesting step-wise accumulation of resistance mutations (*gyrA* T86I) without significant intermediate populations. Human *C. coli* isolates had the highest ciprofloxacin MICs, with 83.3% exceeding the susceptibility breakpoints.

3.4.4. Ampicillin and Amoxicillin-Clavulanate

Ampicillin MIC distributions mirrored the categorical resistance data. All poultry *C. jejuni* and all *C. coli* isolates had MICs of ≥16 mg/L. Amoxicillin-clavulanate MIC distributions were notable for the marked contrast between human *C. jejuni* (91.7% susceptible, MIC ≤ 4 mg/L) and poultry *C. jejuni* (100% resistant, MIC: 16 - 64 mg/L), potentially reflecting differential selective pressure from veterinary *β*-lactam use.

3.5. Multilocus Sequence Typing and Molecular Epidemiology

MLST was successfully assigned to 76 *C. jejuni* (62 human, 14 poultry) and 11 *C. coli* (7 human, 4 poultry) isolates (Table 5). Five *C. jejuni* sequence types were identified, dominated by ST-353 (32 human, 3 poultry; CC-353) and ST-1036 (16 human, 6 poultry; CC-353). ST-1932 (CC-460), identified in 12 human and 2 poultry isolates, demonstrated a broad MDR genotype encompassing *β*-lactams, fluoroquinolones, macrolides, and tetracyclines. ST-2084 (CC-353), identified exclusively in poultry isolates, exhibited an equally broad MDR profile and harboured multiple resistance determinants, including $bla_{\text{OXA-605}}$, *gyr*A T86I conferring resistance to quinolones, 50S ribosomal protein L22 mutation A103V confering resistance to macrolides, and *tet*(O) confering resistance to tetracylines. ST-1036 was notable for its susceptible phenotype across all tested antimicrobials.

C. jejuni strains in the ST-353 (CC-353), ST-2084 (CC-353), and ST-1932 (CC-460) all exhibited fluoroquinolone resistance and carried the *gyrA* T86I mutation that is widespread across both CC-353 and CC-460 lineages (Table 5). Resistance against *β*-lactams such as ampicillin/cefotaxime was observed among isolates belonging to ST-353, ST-2084, ST-1932, and ST-1038/ST-7784 and this resistance was associated with carriage of $bla_{\text{OXA-61}}$ gene that is common across multiple clonal complexes (CC-353, CC-460, and unassigned STs), indicating it is a core or widely disseminated resistance determinant.

Tetracycline resistance in *C. coli*. strains belonging to ST-2084 (CC-353) and ST-1932 (CC-460), as well as ST-830 (CC-828) was associated with the *tet*(O). Macrolide resistance that was observed in strains belonging to ST-2084 (CC-353)

Table 5. MLST types and associated AMR phenotypes and resistance markers.

ST Type	Clonal Complex	Species	AMR Phenotype (Resistance Marker Detected)	Source Distribution
ST-353	CC-353	*C. jejuni*	β-lactams [***bla*$_{OXA-605}$, or *bla*$_{OXA-61}$**] Fluoroquinolones (FQ) [***gyrA* T86**]	Human (32), Poultry (3)
ST-2084	CC-353	*C. jejuni*	β-lactams [***bla*$_{OXA-605}$**], FQ [***gyrA* T86**], Macrolides [*erm*(B)], Tetracycline [*tet*(O)], TMP-SMX [***dfr1.dfr9***]	Poultry (3)
ST-1932	CC-460	*C. jejuni*	β-lactams [***bla*$_{OXA-61}$**], FQ [***gyrA* T86**], Macrolides [**A103V**], Tetracycline [***tet*(O)**]	Human (12), Poultry (2)
ST-1036	CC-353	*C. jejuni*	Tetracycline [***tet*(O)**], TMP-SMX [***dfr9***]	Human (16), Poultry (6)
ST-1038, ST-7784	Not determined	*C. jejuni*	TMP-SMX [***dfr1.dfr9***] β-lactams [***bla*$_{OXA-61}$**]	Human (2)
ST-8043	Related to CC-828	*C. coli*	Macrolides [***gyrA* A103V**], Tetracycline [***tet*(O)**], TMP-SMX [***dfr1***]	Human (3), Poultry (2)
ST-830	CC-828	*C. coli*	Tetracycline [***tet*(O)**], TMP-SMX [***dfr9***]	Poultry (1)
ST-607	CC-460	*C. coli*	Tetracycline [***tet*(O)**] TMP-SMX [***dfr1* or *dfr9***]	Human (3), Poultry (2)

Distribution of multilocus sequence types (STs), clonal complex (CC) assignments, and associated antimicrobial resistance (AMR) phenotypes among *Campylobacter jejuni* (n = 76) and *Campylobacter coli* (n = 11) isolates from human clinical and poultry sources. Species identification was confirmed by multiplex PCR. Sequence types and clonal complexes were assigned using the PubMLST *Campylobacter* database (https://pubmlst.org/campylobacter). AMR phenotype indicates the antimicrobial class(es) to which isolates of each ST demonstrated phenotypic resistance; "Susceptible" denotes susceptibility to all agents tested. Source distribution shows the number of isolates recovered from each host origin. Antimicrobial classes are abbreviated as follows: fluoroquinolones (FQ), trimethoprim-sulfamethoxazole (TMP-SMX), and multidrug-resistant (MDR); defined as non-susceptibility to at least one agent in ≥3 antimicrobial categories, per Magiorakos *et al.* [20]. ST, sequence type; CC, clonal complex; MLST, multilocus sequence typing.

and ST-1932 (CC-460) and those belonging to *C. coli* ST-8043 (related to CC-828) was attributed to carriage of *erm*(B) or 23S rRNA mutations.

Among *C. coli*, three sequence types were identified. ST-8043 (related to CC-828) and ST-607 (CC-460) were both associated with MDR profiles and were recovered from both human and poultry sources. ST-830 (CC-828), found exclusively in a single poultry isolate, demonstrated resistance only to tetracycline. The shared distribution of ST-353, ST-1932, and ST-8043 across human and poultry sources provides molecular evidence of zoonotic circulation between animal reservoirs and human populations.

3.6. MLST and Clonal Diversity

MLST analysis of Kenyan Campylobacter isolates revealed a clear link to global poultry-associated lineages (**Figure 1**). In *C. jejuni*, the predominant STs (ST-353, ST-2084, ST-1036) belong to CC-353, a known chicken-specialist complex, while ST-1932 (CC-460) mirrors prevalent West African types. Notably, ST-1038 and ST-7784 are unassigned, potentially representing novel African lineages. In *C. coli*, ST-8043 (MDR) and ST-830 fall within the dominant CC-828 complex, with ST-830 extending its African distribution eastward. The data show a strong human-

poultry interface, with poultry isolates matching human clinical types, and a total of 62 human *C. jejuni* cases versus 7 *C. coli* cases.

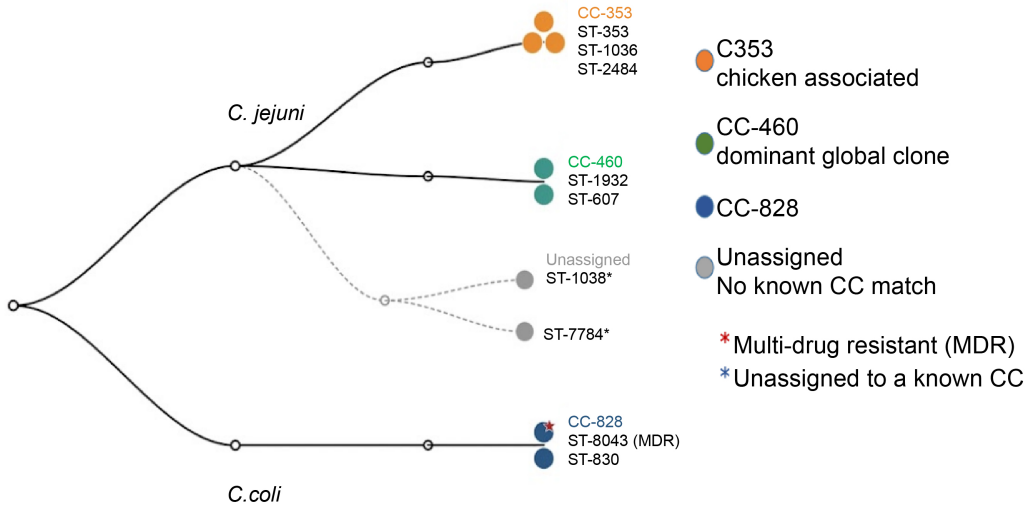

Figure 1. Genetic relationship of locally isolated Camplobacter isolates belonging to various MLST types with those published in other studies. Unrooted neighbour-joining phylogenetic tree depicting the clonal relationships among *Campylobacter jejuni* and *Campylobacter coli* sequence types (STs) identified in this study, constructed from concatenated multilocus sequence typing (MLST) allelic profiles of seven housekeeping loci. Kenyan isolates are positioned in the context of globally recognized clonal complexes (CCs) curated in the PubMLST database (https://pubmlst.org/campylobacter). Node colours denote clonal complex membership: orange, CC-353 (chicken-associated lineage); teal/dark green, CC-460 (dominant global clone); dark navy blue, CC-828 (*C. coli* lineage); grey, STs unassigned to a recognized CC. Node size is proportional to the number of isolates sharing that ST. Dashed branches indicate uncertain phylogenetic placement due to the absence of a formal CC assignment. A red asterisk (*) denotes multidrug-resistant (MDR) status; a blue asterisk (*) denotes STs unassigned to a known clonal complex. The deep bifurcation separating the upper (*C. jejuni*) and lower (*C. coli*) branches reflects the established interspecific divergence within the genus *Campylobacter*. ST, sequence type; CC, clonal complex; MDR, multidrug-resistant.

4. Discussion

4.1. Prevalence and Risk Factors

The Campylobacter isolation rate of 11.6%, with *C. jejuni* predominance (89.2%), is consistent with sub-Saharan African hospital-based surveillance reporting pooled prevalence of 9.9% (CI: 8.4% - 11.6%) and *C. jejuni* as the most prevalent species [2] [3]. The highest isolation in children under two years (13.7%) reflects immunological immaturity compounded by frequent faecal-oral exposures in low-resource settings, where community-based studies demonstrate that up to 60,000 per 100,000 children under five are affected [41]. Homestead poultry farming emerged as the strongest risk factor across all age strata (OR: 6.47 - 10.05), implicating the domestic poultry environment as the primary transmission reservoir; a finding corroborated by the shared MLST genotypes between human and poultry isolates in this study and consistent with broader evidence that colonized broiler chicks may be the primary vector for transmitting Campylobacter to humans [42] [43]. Full risk factor analyses are detailed in Ouko *et al.* [1].

4.2. Antimicrobial Resistance

Universal resistance to tetracycline (100%) and trimethoprim-sulfamethoxazole (95% - 100%) confirms resistance saturation for these agents in this setting, rendering them clinically obsolete for treating this pathogen. This pattern is well-documented across sub-Saharan Africa and is partially attributable to decades of unregulated use in human and veterinary medicine [3] [44] with global antimicrobial consumption in livestock estimated at 63,151 tons in 2010. These volumes are projected to rise by 67% by 2030, with chicken production consuming 148 $mg \cdot kg^{-1}$ of antimicrobials annually [45].

Erythromycin resistance in human *C. jejuni* (57.1%) reported in the current study substantially exceeds rates in Western Europe (<5%) and North America (3% - 10%) [46]. This scenario probably represents a critical therapeutic concern given that macrolides remain the drugs of choice for severe campylobacteriosis following increasing fluoroquinolone resistance [6]. The absence of erythromycin resistance in poultry *C. jejuni* observed in this study suggests macrolide selection pressure operates predominantly at the human medicine interface, or that resistant strains originate from unsampled reservoirs; a hypothesis requiring whole-genome sequencing and source attribution modeling to resolve.

Ciprofloxacin resistance in human *C. jejuni* (36.9%) and *C. coli* (80.0%) observed in the current study aligns with global escalation trends following fluoroquinolone introduction in poultry production [47]. Third-generation cephalosporin resistance in *C. coli* (ceftriaxone 80% - 100%; cefotaxime 60% - 75%) is consistent with emerging extended-spectrum β-lactamase-like patterns reported from Ethiopia and Nigeria [44]. This finding is particularly alarming as these agents serve as reserve antibiotics for severe paediatric infections in resource-limited settings.

The classification of human *C. coli* as extensively drug-resistant (XDR), resistant to 10 of 11 antibiotics across all six classes tested, represents a grave public health threat. XDR Campylobacter may necessitate the use of carbapenems or tigecycline, agents rarely accessible or affordable in sub-Saharan African clinical settings. Similar Multidrug-resistant Campylobacter outbreaks with resistance to all commonly used antibiotics have been documented in the United States linked to animal reservoirs [10]. The CDC estimates that over 300,000 drug-resistant Campylobacter infections are reported annually [46].

4.3. Molecular Epidemiology of Resistance Determinants

Fluoroquinolone resistance across *C. jejuni* ST-353 (CC-353), ST-2084 (CC-353), and ST-1932 (CC-460) was primarily mediated by the *gyrA* T86I mutation, which shows 100% concordance with phenotypic ciprofloxacin resistance [7] [48]. Its distribution across two distinct clonal complexes indicates independent selection events rather than clonal expansion alone. The T86I substitution additionally confers a fitness advantage, explaining its persistence without ongoing antibiotic pressure [49].

β-lactam resistance in ST-353, ST-2084, ST-1932, and ST-1038/ST-7784 was largely associated with $bla_{\text{OXA-61}}$, a class D β-lactamase carried by 91% of ampicillin-resistant campylobacters globally [32]. The $bla_{\text{OXA-605}}$ also detected in some isolates is now considered a variant of $bla_{\text{OXA-61}}$ conferring almost an identical resistance phenotype. Its detection across CC-353, CC-460, and unassigned STs confirms $bla_{\text{OXA-61}}$-like β-lactamass may be the core resistance determinants whose expression is modulated by a promoter single-nucleotide polymorphism (SNP). This differential expression accounts for variable phenotypic resistance levels observed in different strains [31] [50]. Higher prevalence of $bla_{\text{OXA-61}}$ in chicken faeces compared with human clinical isolates supports the poultry as possible reservoir of strains harboring these β-lactam resistance genes [51].

Tetracycline resistance in various strains largely affiliated to the ST-2084 (CC-353), ST-1932 (CC-460), and *C. coli* ST-830 (CC-828) was mediated by the plasmid-borne *tet*(O) gene, encoding a ribosomal protection protein originally characterized on conjugative plasmids in *C. jejuni* [52] [53]. Its presence across phylogenetically divergent lineages and both species suggests horizontal gene transfer via conjugative plasmids, including, as reported in previous studies, megaplasmids carrying Type IV secretion systems, as the primary dissemination mechanism [54] [55]. Mosaic variants such as *tet*(O/M/O) further expand the diversity of tetracycline resistance determinants [56]. This explains the universal tetracycline resistance observed phenotypically, as *tet*(O)-bearing plasmids spread independently of clonal background [9].

Macrolide resistance in most strains, especially those belonging to ST-2084 (CC-353), ST-1932 (CC-460), and *C. coli* ST-8043 (CC-828) was attributed to *erm*(B) or 23S rRNA mutations (A2075G/A2074T). The *erm*(B) gene has been reported to reside on transferable multidrug resistance genomic islands (MDRGIs) of Gram-positive origin and is consistently co-located with determinants conferring resistance to multiple drug classes [8]. Horizontal transfer of *erm*(B) via natural transformation has been confirmed experimentally and epidemiologically, with recent genomic studies identifying horizontal gene transfer as the predominant mechanism driving *erm*(B) dissemination across diverse ecological reservoirs [57]. The 23S rRNA A2075G substitution and *erm*(B) have been reported in 4.2% and 4.9% of isolates respectively in central China, correlating with erythromycin resistance [58]. Critically, ST-1036 (CC-353) remained fully susceptible, demonstrating that resistance within CC-353 reflects differential gene acquisition at the sequence type level rather than lineage-intrinsic properties.

4.4. Potential Zoonotic Transmission and Clonal Dynamics

The dominance of CC-353 and CC-460 in both human and poultry isolates, with shared sequence types ST-353, ST-1932, and ST-8043 recovered from both sources, strongly suggests a potential zoonotic transmission pathway between humans and poultry. The ST-353 (CC-353) reported in this study is a globally disseminated poultry-associated genotype consistently identified in source attribution studies

and recently shown to cause localized gut inflammation and extra-intestinal spread in broiler chickens [15] [59]. Other strains belonging to ST-1932 (CC-460), carrying a four-class MDR profile in both hosts, raise concern about a possible active dissemination of multiply resistant lineages through the food chain. These findings are consistent with other studies that have confirmed shared population structures between human and poultry *Campylobacter* isolates in the region [60].

Based on our current findings, the ST-2084 (CC-353) is apparently restricted to poultry with the most complete resistance gene repertoire ($bla_{\text{OXA-61}}$, *gyrA* T86I, L22 A103V, *tet*(O)), which may represent early-stage reservoir colonization with potential for subsequent human transmission. ST-1038 and ST-7784, unassigned to established clonal complexes, potentially represent novel African lineages not yet captured in global databases, warranting expanded whole-genome sequencing and integration into international MLST surveillance networks [13] [15] [36].

4.5. Policy Implications

These findings strongly suggest the need for: 1) removal of tetracycline and trimethoprim-sulfamethoxazole from empirical campylobacteriosis treatment guidelines, with updated protocols reflecting high fluoroquinolone and macrolide resistance; 2) urgent escalation of AMR surveillance using standardised methodologies, as recommended by WHO's designation of fluoroquinolone-resistant *Campylobacter* as a high-priority pathogen [11]; 3) targeted One Health interventions; hygienic poultry husbandry, restricted veterinary antimicrobial access, household education on food safety, and reinforced WASH infrastructure; and 4) integrated genomic surveillance platforms linking clinical, veterinary, and food safety laboratories to monitor resistance gene flow across the livestock-human interface [61] [62].

4.6. Limitations

The small poultry *C. coli* sample (n = 4) limits source-specific inference while some isolates recovered in the original study were lost to culture. The cross-sectional design precludes causal conclusions, and self-reported exposures are subject to recall bias. Furthermore, some isolates could not be assigned to established sequence types, warranting a deeper molecular analysis using advanced techniques such as WGS. Non-poultry animal reservoirs were not assessed, limiting source attribution resolution.

5. Conclusion

This study documents substantial *Campylobacter* AMR burden and molecular evidence of zoonotic transmission in western Kenya. Universal resistance to first-line agents, XDR *C. coli* emergence, and shared MDR genotypes circulating between poultry and humans collectively demand urgent One Health responses integrating antimicrobial stewardship, enhanced surveillance, and targeted interven-

tions at the livestock-human interface.

Data Availability

The datasets used or analyzed during the current study are available from the corresponding author upon request.

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Author Contributions

TO was responsible for the project design, field and laboratory sampling, and manuscript preparation. SK and ANK contributed supervisory oversight and assisted with manuscript development. EMF secured funding through the ESEI project, contributed to the study design, and assisted in manuscript preparation. JMN provided laboratory support and contributed to the writing of the manuscript. JSK edited the tables, validated the statistics and revised the final version. MMK performed data analysis and prepared the manuscript tables and figures. JK offered supervisory guidance, supported molecular analysis, and participated in manuscript preparation and revisions.

Declarations

This work was approved by KEMRI independent Scientific and Ethical Research Unit (SERU) under approval number *27/09/2016_3320*. Standard guidelines for recruitment and ethical access of literature cited were adhered to. No animals were used or harmed during the course of this study.

Conflicts of Interest

All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this manuscript.

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